DISSERTATION ON
"A COMPREHENSIVE STUDY OF 110 CASES OF LIVER ABSCESS"

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CERTIFICATE

This is to certify that this dissertation is a bonafide work of **Dr. R. BALAMURUGAN** on **"A COMPREHENSIVE STUDY OF 110 CASES OF LIVER ABSCESS"** in Department of General Surgery during the years 2003-2006 in **KILPAUK MEDICAL COLLEGE & HOSPITAL, CHENNAI – 600 010.**

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CONTENTS

INTRODUCTION

Descriptions of Liver abscesses date back to Hippocrates in approximately 4000 BC. But an understanding of their Etiology, Bacteriology, diagnosis and treatment is a recent event in Twentieth Century and is still emerging.

Hepatic abscess often presents a pitfall in diagnosis and challenge to surgical diagnostic acumen. Early diagnosis and prompt initiation of treatment almost certainly leads to complete cure.

Of the two types of hepatic abscess, the Amoebic and Pyogenic, the former seems to be more prevalent in our country. Because studies of Hepatic abscess especially amoebic abscess have often originated from endemic areas, they offer little information on the application of modern diagnostic techniques. Diagnosis in this area mainly depends on the clinical presentation. But since the introduction of Ultra sonogram as a diagnostic device a more Accurate Diagnosis can be made in every case.

In this study, 110 cases of Hepatic abscesses admitted in Government Kilpauk Hospital in the year 2003-2006 are analysed and discussed.

HISTORICAL FACTS

JOHN BRIGHT	1836	First Description of the Hepatic abscess in Modern Medicine.
WALLER & LATER DIEULAFOY		Described the Association of Hepatic Abscess (Pyogenic) in patients with Appendicitis and Pyle phlebitis.
LAMBL	1859	Discovered the Parasite Entamoeba Histolytica.
LOSCH	1875	Proved the pathogenic nature of E. Histolytica.
KARTULIS	1887	Described the Presence of E. Histolytica In Liver abscess
SIR WILLIAM OSLER	1890	First reported the Coexistence of Hepatic and Colonic Amoebiasis
COUNCILMAN AND LAFLEUR	1891	Coined the term Amoebic abscess of Liver
CRAIC	1927	Defined the Modern Concept of Clinical Amoebiasis
OCHSNER AND ASSOCIATES	1938	Collected Review of Pyogenic Liver Abscess in the Pre antibiotic era.

AIMS AND OBJECTIVES

Clinically impressed with the great importance of establishing early diagnosis in patients with liver abscess, an Endeavour has been made in this study to obtain better knowledge of the nature of the disease.

* To determine the incidence of Hepatic abscess in our Hospital.

* To determine the Age and Sex incidence.

* To evaluate the various clinical parameters of both Uncomplicated and complicated Liver abscess and various modalities of treatment available.

* To throw light on the disease Prevalence occurring as Abdominal Emergencies.

* To emphasis the immense use of sophisticated modern Investigations especially Ultra sonogram in the confirmation of diagnosis as well as in the treatment and follow up.

ANATOMY OF THE LIVER

The liver is the largest gland in the body. It is situated in the upper and right parts of an abdominal cavity, occupying almost the whole of the right hypochondrium, greater part of epigastrium, extending into left hypochondrium upto left lateral line. In male it weighs about 1400-1800g and 1200-1400g in female.

It has 5 surfaces. (Anterior, posterior, superior, inferior and the right). One prominent border (Inferior Border). On the posterior surface there lies the **"Bare area of liver"** which is contained within the reflection of superior and inferior coronary ligaments. Liver abscess is commonly situated in the upper part of the right lobe close to the Bare area. Liver abscess close to the bare area is painless because they are devoid of peritoneum.

Surface:

Diaphragmatic Surface:

a. Anterior

b. Superior

c. Posterior

Visceral Surface:

a. Postero Inferior

Diaphragmatic Surface:

Smooth and doom shape, related to concavity of inferior surface of diaphragm, separated from diaphragm by sub-phrenic recesses which is separated by falciform ligament into Right and Left.

The convex or antero superior surface of liver is in relation with right lung and the pleura. This explains the erosion of hepatic abscess through the diaphragm into the pleural cavity and right lung.

PERITONEAL LIGAMENTS OF THE LIVER

a. Falciform ligament
b. Superior and inferior coronary ligament
c. Right Triangular ligament
d. Left Triangular ligament
e. Lesser omentum

a. Falciform ligament

The liver is connected to the anterior abdominal wall and the diaphragm by falciform ligament, the lower free border of which is termed the ligamentum teres and contains obliterated left umbilical vein.

b. Superior and Inferior Coronary ligament

Reflection of peritoneum from diaphragm to bare area, anteriorly, superior coronary ligament, posteriorly, inferior coronary ligament.

c. Right Triangular ligament

Two layers of coronary ligament meet on right to form Right Coronary Ligament.

d. Left Triangular Ligament:

The left layers of falciform ligament and lesser omentum meet to form left triangular ligament.

e. Lesser omentum:

Enclosing the portal triad passes from liver to lesser curvature of stomach and first 2 cms. of first part of duodenum. Thick free edge of lesser omentum extending between porta hepatis and duodenum is Hepato duodenal ligament and the sheet of ligament extending from ligamentum venosum and lesser curvature of stomach is Hepato Gastric Ligament.

PORTA HEPATIS

Is a deep transverse fissure about 2 inch long, situated on the inferior surface of the right lobe. It admits portal vein, Hepatic Artery and the Hepatic plexus of Nerves and lets out the right and left Hepatic ducts and

few lymphatics. Relations within the Porta Hepatis are from behind far wards – the portal vein, hepatic a, and Bile ducts.

MORPHOLOGICAL ANATOMY

Liver is divided into the right and left lobes by the falciform ligament anteriorly and superiorly, by the fissure for ligamentum teres inferiorly and by the fissure for ligamentum venosum posteriorly. Right lobe is much larger and forms 5/6 part. It has 2 additional lobes called the caudate and quadrate lobe.

The caudate (SPIGEL) Lobe: 4^{th} lobe of liver is delineated by the ligament of **Aranticus** to the left, posterior to transverse fissure of the hilus. The left lobe of liver forms only 1/6 part of liver.

FUNCTIONAL ANATOMY OR SURGICAL ANATOMY OR SEGMENTAL ANATOMY

Functional anatomy was first initiated by cantle in 1898 and was enhanced by Counseller in 1929 and **Couinaud** in 1957. **Couinaud's** is the most exact and complete description of liver anatomy.

Liver is divided into 2 lobes or hemi liver by main portal fissure or scissura called Cantle's line. Main Portal fissure describes a 75 angle with

horizontal plane and extends from the antero inferior gall bladder fossa, postero superiorly to the left side of inferior venacavae.

The left lobe of liver consists of Hepatic tissues to the left of falciform ligament plus the quadrate lobe and caudate lobe of the old anatomic nomenclature. The right Hepatic lobe contains remaining Hepatic tissues.

The right portal fissure divides the right lobe into an antero medial and postero lateral sector. The right Hepatic vein courses along this fissure. The left portal fissure divides the left lobe into anterior and posterior sector and it is in this fissure, the left hepatic vein courses. The left portal fissure located posteriorly in relation to ligamentum teres. The spigelian lobe or segment 1 is considered as an Autonomous segment.

HEPATIC STRUCTURE

The greater part of the liver is invested with peritoneum, which covers a thin capsule of connective tissue (Glisson's capsule). The bulk of the cells within the liver constituting about 80% are the hepatocyctes or parenchymal cells. Hepatocytes carry out a multitude of metabolic activities. **Hepatic lobule** consists of a central vein as its central axis and surrounded at its edges by groups of 3 tubes each being termed a portal

triad. **Portal lobule** consists of the adjoining parts of the hepatic lobules, the bile from which drains into a Bile ductule in the portal canal at the meeting place of the 3 hepatic lobules.

Hepatic macrophages or stellate cells of von kupffer (Kupffer) cells. These line the interlobular venous sinusoids and from a major part of the mononuclear phagocyte system. The sinusoids lie in the hepatic lacunae between the hepatic laminae and are separated from the liver plates by the **"Perisinusoidal Space of Disse".**

BLOOD SUPPLY

The dual afferent blood supply consists of hepatic artery and portal vein. Liver receives 20% of its blood supply from hepatic artery and 80% from the portal vein. Before entering the liver the hepatic Artery and portal vein divide into right and left branches. Within the liver they redivide to form the segmental and then to interlobular vessels which run in the portal canals. Further ramification of interlobular branches open into the hepatic sinusoids. Thus hepatic arterial blood mixes with the portal venous blood in the sinusoids.

VENOUS DRAINAGE

The hepatic venous drainage beings as a central vein of a liver lobule. The Central vein receives sinusoids from all sides and unite with the central vein of other lobules to form the sub lobular veins which in turn fuse to form collecting veins which forms 3 major hepatic veins.

LYMPHATIC DRAINAGE

The superficial lymphatics drain into caval, hepatic, paracardial and coeliac lymph nodes. Deep lymphatic end in the nodes around the end of IVC and partly into hepatic nodes.

NERVE SUPPLY

Nerves of liver are derived from Hepatic Nerve plexus which are largest derivative of coeliac plexus. Hepatic plexus accompanies branches of Hepatic artery and portal vein to the liver. It consists of sympathetic fibres from coeliac plexus and para sympathetic fibres from anterior and posterior vagal trunks.

DEVELOPMENT

The liver arises from Hepatic diverticulum from most caudal part of the foregut. The Hepatic diverticulum extends into septum transversum and expands the ventral mesentry. It divides into a large cranial part which give

rise to interlacing cords of liver cells and intra epithelial lining of intra Hepatic portion of biliary apparatus.

FUNCTIONS OF THE LIVER

1. The formation of bile and the metabolism of bilirubin and of bile salts.
2. The synthesis of albumin, fibrinogen and prothrombin.
3. Storage and metabolism of carbohydrates, including the conversion of monosaccharides (e.g. dextrose) into glycogen, and vice versa.
4. Formation of phospholipids and cholesterol, synthesis of fatty acids from carbohydrate.
5. Deamination of amino acids with formation of urea. Removal of ammonia from portal blood.
6. Heat production
7. Reticuloendothelial activities
8. Storage of Vitamin B_{12} and Vitamin A
9. Iron and Copper storage
10. Destruction of bacteria
11. Detoxication of drugs and hormones.

REVIEW OF LITERATURE

INCIDENCE

Entamoeba histolytica Infection affects an estimated 10% of the world's population. The great majority of such Infections occurring in people living in Indian sub-continent, sub Saharan Africa, and parts of Central and South America. In these endemic areas approximately 50% of the population is infected with 90% or more being Asymptomatic cyst Passers. Amoebic Liver abscess occurs in less than 10% of individuals infected with these organisms. Amoebic Liver abscess are 3-5 times more frequent than Pyogenic Liver abscess. Average age of the patients is between 28-48 years, with striking male predominance (20:1). Particularly severe Invasive disease occurs in patients with compromised cellular immunity in young infants, in the malnourished, in pregnant women and in patients receiving corticosteroid. On the global scale, Amoebiasis is the third most common Parasitic cause of Death after malaria and schistosomiasis.

Incidence of Pyogenic Liver abscess being estimated at 8-16 cases / 1,00,000 admissions. Fifty years ago the majority of patients were under the age of 40 and Appendicitis was the leading cause of the disease. Today the average age being 43-60. This change corresponds to the finding that

appendicitis has been replaced by Biliary tract disease as the most common underlying aetiology.

LIFE CYCLE OF E-HISTOLYTICA

Amoebiasis is defined as the condition of Harboring of entamoeba Histolytica in Humans with or without clinical Manifestation (WHO Tehran in 1968)

E.Histolytica exists in two forms

1. Trophozoite
2. Cyst with a Transitory stage of Precystic Form.

E.Histolytica Passes its life cycle only in one Host the man.

The mature quadrinucleate cysts are the infective forms of the parasite. When these cysts are swallowed along with contaminated food and drinking water by a susceptible person, they are capable of further development inside his gut. The fully developed cysts thus gaining entrance in to the alimentary canal, pass unaltered through the stomach. The “Excystation” occurs when the cyst reaches the caecum or lower part of the ileum. Each cyst Liberates a single Amoeba with four Nuclei, a tetranucleate Amoeba which eventually forms eight Amoebulae (Metacystic trophozoites) by division of nuclei with successive fission of

cytoplasm. The young Amoebulae being actively motile, invade the tissues and ultimately lodge in the submucous tissue of the large gut their normal habitat. Here they grow and multiply by Binary fission.

It is to be noted that the Trophozoite phase of the parasite is responsible for producing the characteristic lesion of amoebiasis. The Trophozoises of E.Histolytica enter into the deeper layers and many find their way into the portal vein, to be carried away to the liver where their further progress may be arrested. In the liver the Trophic forms may for a time grow and multiply but encystations does not occur.

Hence such an invasion is always to be overlooked as an accident on the part of the Parasite because so far as its biological aspect is concerned it has reached a dead end. The parasites that remain in the intestinal wall may cause an attack of acute dysentery (amoebic colitis). A certain number of these Trophozoites are discharged into the lumen of the Bowel and are transformed into small precystic forms from which the cysts develop.

The mature quadrinucleate cysts are the most resistant and infective forms of the parasite. But the cysts produced in an infected individual are unable to develop in the host in which they are produced. Transfer to another susceptible host enables them to grow and continue their life cycle.

There are 2 types of E.histolytica namely pathogenic and non pathogenic. It is now firmly believed that pathogenic and non-pathogenic entamoeba isolates are distinct species. Pathogenic is called E.Histolytica, Non pathogenic as **E.Dispar.** Pathogenic behaviour of pathogenic and non pathogenic entamoeba depends on the quantitative and qualitative difference between molecules (genes/proteins) indentifiable in both forms (Indian journal of gastroenterology – April 1998) Volume : 17 p.58.

AETIOLOGY AND PATHOGENESIS – AMOEBIC LIVER ABSCESS (Syn TROPICAL OR DYSENTERIC ABSCESS)

The transmission of amoebiasis is clearly related to lack of sanitation and low socio-economic status rather than to climate. Common modes of transmission are by food contaminated with cysts or contaminated water through those engaged in preparation and handling of food. The commonest extra intestinal manifestation of amoebic infection occurs when organisms escape the colon and reach the liver via the portal vein.

There is another view that retrograde lymphatic spread of Amoebiasis can occur from the colon to the liver and from the liver to the lungs. Lymphangiographic study in case of carcinoma stomach have shown that the lymphatics from the stomach, colon and liver join the thoracic duct – an evidence for the retrograde lymphatic spread of amoebiasis. It was favored by the demonstration of multiple amoebae in the small interlobular

branches of the portal vein. After reaching the liver in large number, the trophozoites set up a thrombus formation which in turn gives raise to an infarct, the wall of the vessel is destroyed and amoebae feed on the products of cytolysis. This view was supported by the demonstration of thrombosed radicals of the portal vein in the walls of the abscess and amoebae were found entangled in the thrombus.

E-histolytica produces enzymes which cause hydrolytic dissolution of liver tissue. Various enzymes are produced like hyaluronidase, Glutaminase, amylase, maltase, esterase, succinyl dehydrogenase and gelatinase. Alcoholism, nutrition particularly poor in protein and poor immune status predispose to the development of amoebic liver abscess.

E. Histolytica-	Adherence to cell by Cal.N.AC. Inhibitable Adhesion ---------->	Cytolosis of cell. Phagocytosis of dead Target cell by Amoeba.

PATHOGENIC CLASSIFICATION OF AMOEBIC LIVER ABSCESS

(Gasto Enterology Today Vol.I No.3)

	Acute	Chronic
a.	Benign	a. Benign
b.	Aggressive	b. Acclerated
1.	Illness less than 10 days	Low Grade symptom for a month or more
2.	Fever & Tender Hepatomegaly present	Fever uncommon, mildly Tender hepatomegaly
3.	Low Serological titres Leukocytosis, increase transminase	High Serological titres, severe anemia ↑ alkaline phosphatase, normal SGOT.

MACROSCOPIC PATHOLOGY

About 83% of liver abscess are located in the right postero-superior surface of liver. The Propensity for this site reflects the fact that venous return from the right side of colon (Amoebic infection having a particular impact on the caecum and Right colon) in to the portal vein is predominantly delivered to the right lobe of liver. To the naked eye, the appearance of the abscess area is reddish brown in color with a semifluid or grumous consistency. The wall of the abscess cavity is ragged and shaggy

in appearance and is formed by the necrotic liver tissue which gradually merges into the healthy zones with an intervening zone of hyperaemia. In an old abscess the wall is smooth and is formed by dense connective tissues. Multiple small abscesses in which the whole organ becomes riddled with scattered foci of necrosis are probably more common. The liver enlargement is explained on the basis of oedema and congestion **(lamot and pooler 1958).**

MICROSCOPY

If a section is made through the margin of a liver abscess, three zones can be differentiated from the center to the periphery.

1. A central zone of cytolysed granular material with no amoebae.
2. An intermediate zone consisting of degenerated liver cells, a few leucocytes, connective tissue cells, red blood cells and an occasional trophozoite of E.histolytica.
3. A peripheral zone consisting of congested capillaries with varying degrees of necrosis of liver cells. The amoebae can be seen to be multiplying in this area and invading and adjoining healthy liver tissue.

In long standing cases, the third zone may consist of actively proliferating connective tissue cells, lymphocytes and monocytes, walling off the abscess cavity.

PUS OF LIVER ABSCESS

The ‘pus’ is not of suppuration but is a mixture of sloughed liver tissue and blood. It is chocolate brown in color and thick in consistency (so called ‘Anchovy-sauce’ pus). The smell is rarely offensive. The ‘pus’ is bacteriologically sterile. Microscopy reveals degenerated liver cells, a few red blood cells and occasional leucocytes. The trophozoites of E.histolytic are not generally found in freshly aspirated pus but appear in the escaping “pus” four or five days after the initial evacuation.

INTESTINAL LESIONS IN AMOEBIC LIVER ABSCESS

In all cases of hepatic amoebiasis, as also in other metastatic amoebiasis, the primary lesion is in the large gut. The changes in the intestine observed at autopsy may be any one of the following.

i) Small superficial ulcers, with thickening of the colon.
ii) A single latent ulcer, located most commonly in the caecum.
iii) Pigmented or non-pigmented scars in the large intestine, representing the sites of previous ulcers.
iv) No change in the large gut.

v) Extensive ulcers scattered throughout the large intestine are quite uncommon.

vi) E-Histolytica can produce diffuse inflammation and ulcertation of colour which may be indistinguishable endoscopically from idiopathic ulcerative colitis. Further more reappearance of colonic symptoms in patients with chronic ulcerative colitis has at times found to be due to infection with E.Histolytica.

PYOGENIC LIVER ABSCESS

ETIOLOGY – BACTERIOLOGY – PATHOGENESIS

In the liver the kupffer cell functions as a primary Barrier and filter for the clearance of micro organisms from arterial, venous, Biliary and local sources. Pyogenic liver abscess are believed to occur when their normal hepatic clearance fails or is overwhelmed. Distal infectious sites may seed the liver with pathogenic Bacteria via portal vein or the Hepatic Artery.

ORGANISMS

Gram Negative (50-70%)

E.Coil (35-45%), klebsiella, Proteus, enterobacter.

Gram Positive (30%)

Streptococcal Sp, enterococcus Faecalis.

Anaerobes (40-50%)

Bacteroides Sp, Fusobacterium, Pepto Streptococcus.

Fungal

Sterile

ETIOLOGY	%	SOURCE	DISTRIBUTION	1° MICRO ORGANISMS
Biliary System	40	Cholangitis Biliary Obstruction	Both Lobes, Multiple	Single Species Gram-aerobes and anaerobes – E-Coli
Portal Circulation	20	Intra abdominal Infection Liver metastasis	Right lobe > Left Multiple or single Area of Metastasis	E. Faecalis, E.Coli, B. Fragilis B. Fragilis
Arterial Circulation	12	Bacteremia, Systemmic Infection	Both lobes- Multiple	S. Aureus, S. Pyogenes
Trauma	4	Direct Exposure, Necrosis	Area of injury	S. Aureus, S. Pyogenes
Direct extension	6	Cholecystitis Perforated ulcer	Adjacent area	E.Coli
Cryptogenic		Unknown	Right lobe > left	B. Fragilis

CLINICAL PRESENTATION

The clinical feature often reflects the site, size and the number of abscesses as well as degree of involvement of adjacent tissues. The onset of symptoms may be acute or chronic. The clinical features of amoebic abscess are similar to Pyogenic abscess.

However acute presentations are more common with amoebic abscess. In Amoebic abscess pain in Right Hypochondrium and Right Lower Chest are the commonest symptom (90%) followed by fever. In Pyogenic abscess, fever is the most common symptom (80%). Hepatomegaly and Right upper Quadrant tenderness are the only consistent Physical findings.

Triad of fever, pain in Right Hypochondrium and Tender Hepatomegaly is strongly suggestive of liver abscess.

Co-morbid diseases associated with Pyogenic liver abscess.

Children	Adults
Chronic granulomatous disease	Diabetes Mellitus
Complement deficiencies	Cirrhosis
Leukemia	Chronic Pancreatitis
Malignancy	Peptic ulcer disease
Sickle cell anaemia	Inflammatory Bowel disease
Polycystic liver disease	Jaundice
Congenital hepatic fibrosis	Tuberculosis
Post transplant liver failure	Malignancy
Necrotising enterocolitis	Leukemia and Lymphoma
Chemotherapy and steroid therapy	Chemotherapy and steroid therapy
Acquired Immuno Deficiency Syndrome	Acquired Immuno Deficiency Syndrome

SYMPTOMS	SIGNS
Pain	Hepatomegaly
Fever	Right upper Quadrant Tenderness
Nausea and Vomiting	Guarding and rigidity
Weightloss	Ascites
Malaise	Jaundice
Diarrhoea	Pleural effusion
Pruritus	Pleural rub
Cough	

AMOEBIC	PYOGENIC
Age<50 years	>50 years
Male: Female 10:1	Male = Female
Recent Travel to endemic area	Malignancy
Pulmonary dysfunction	High fevers
Abdominal pain	Pruritus
Diarrhoea	Jaundice
Abdominal Tenderness	Septic shock

Left Lobe Abscess

Deserves special consideration as the diagnosis is delayed or missed resulting in more complications and a higher mortality. Proximity to Pericardium, propensity to rupture easily makes early diagnosis and treatment important.

COMPLICATIONS OF LIVER ABSCESS

About 40% of patients with Pyogenic liver abscess and 10% of amoebic liver abscess patients develop complications. Generalized sepsis is the most common systemic complication in Pyogenic abscess. Rupture into peritoneum and thorax are the most common complications in amoebic abscess. Posteriorly located Amoebic Liver Abscess in Right Lobe can

present as IVC obstruction or Hepatic out flow obstruction (Gastro Enterology Today April, June 1997).

Severe Amoebic Liver Abscess (Chau.Sk., Chang. Chiencs, Sheen IS)

Severe Amoebic Liver Abscess was defined as the Rupture of an abscess that was resistant to 72 Hours of medical treatment, complicated by secondary bacterial infection or Diabetes Mellitus.

PLEUROPULMONARY COMPLICATION

It is the most frequent site affected when liver abscess ruptures. It is the third most common manifestation of amoebiasis after amoebic colitis and amoebic liver abscess. The reported incidence of pleuropulmonary amoebiasis varied from 14 to 30 percent. Clinical manifestations include chest Symptoms with expectoration of anchovy – sauce sputum. Pleural rub, signs of pleural effusion and lung abscess may also be present. Typically such lesions originate from right lobe.

PERITONEAL COMPLICATION

Amoebic peritonitis is the second most common complication of Amoebic liver abscess and it is the commonest form of rupture into a serous cavity. Amoebic peritonitis results more often due to rupture of

Right Lobe Abscess. Extension into Peritoneal cavity may form acute generalized peritonitis, chronic generalized peritonitis or a localized abscess. In sudden rupture of amoebic liver abscess the clinical picture mimics perforation of a hollow viscus. Mortality may approach a great as 20%.

PERICARDIAL COMPLICATION

Pericardial complications are relatively rare and are often associated with left lobe abscess. The reported incidence of this complication varies from 0.6 to 5 percent. Pericardial extension may cause suppurative pericarditis due to sudden rupture of a abscess in to the pericardial cavity. Non-purulent type may present as pericardial effusion. It can also cause constrictive amoebic pericarditis or amoebic hydro pericardium.

UNCOMMON COMPLICATIONS

Erosion into the intestinal tract, cutaneous fistulation, secondary infection, obstructive jaundice, subphrenic abscess, hemobilia and vascular complications are other rare complications. Siddiqui MN, Rizvi SB, Ahmed M, in their case report, reported a case of Amoebic Liver Abscess complicated by Hepato duodenal fistula (a finding of air in the liver abscess – on Ultra sonogram – confirmed by Gastograffin Swallow)

DIAGNOSTIC STUDIES

Laboratory investigations

Even though most of the laboratory investigations are non specific, they are helpful to some extent in the diagnosis, to assess the involvement of liver, response to treatment and also as prognostic factor.

BLOOD

* **Leucocytosis** varying from 15,000–25,000 with a left shift present in 50-60%
* **Anemia** is also encounted in liver abscess. It is related to duration and size of Abscess.
* **Erythrocyte sedimentation rate** is also usually raised.

LIVER FUNCTION TESTS

Abnormal liver function test exist more commonly in Pyogenic liver abscess than Amoebic Liver Abscess.

SERUM BILIRUBIN

* Serum bilirubin is elevated in 10-24% of patients.

ENZYMES

* Serum Alkaline phosphatase is raised in about 50-60% of patients.
* Elevations of SGOT or SGPT and Gamma – Glutamyl Transpeptidase occur in approximately 40-50% of patients.

SERIUM PROTEINS

* Hypoalbuminemia is quite common with increase in Gamma – Globulin.
* Hypoalbuminemia is considered as a poor prognostic marker especially if it is < 2gm/dl

PROTHROMBIN LEVEL

* There is also prolonged plasma prothrombin level.

PARASITOLOGICAL EXAMINATION

STOOL EXAMINATION

Cyst & Trophozoites of Entamoeba Histolytica are present in the stools in <15% of Amoebic liver abscess patients – verlenden et al. This gives information regarding the persistence of intestinal infection.

ASPIRATE

Aspirate is odourless gram stain and culture are negative in amoebic liver abscess. But we can culture the organisms in case of Pyogenic

abscess. Recovery of trophozoites of Entamoeba Histolytica in the PUS establishes the diagnosis of amoebic liver abscess. Addition of wall scrapping containing viable trophozoites can increase the sensitivity of Aspirate.

IMMUNO DIAGNOSTIC METHODS

These tests are useful in cases of Amoebic aetiology

INDIRECT HAEMAGGLUTINATION TEST (IHA)

* 95% Sensitive – pahuchonma, patterson et al.

  Positive if dilutions exceed 1:128

  Highly specific for invasive amoebic disease

  Remains positive for 20 years.

GEL DIFFUSION PRECIPITIN TEST (GDP)

* 90-95% Sensitive. Detects non-invasive amoebic colitis as well.

  So it is sensitive and non specific test. Remains positive for 6 months.

OTHER TESTS USED RARELY

1. Enzyme Linked ImmunoSorbent Assay (ELISA)

   Amoebic Antibodies in Titres >1: 400 is considered as evidence of Amoebic Liver abscess.

2. Immuno Electrophoresis (IE)
3. Counter Current Electrophoresis (CEP)
4. Immuno – Fluorescent Antibody (IF) Test
5. Latex agglutination (LA) Test
6. Radio Immuno Assay (RIA) Test
7. Compliment Fixation Test (CF)
8. Intradermal Test
9. Immobilization Test
10. Specific DNA probes:

* Specific DNA probes for pathogenic strain is 145 Base pair multi copy gene. For non pathogenic strain is a 133 Base Pair multicopy – DNA fragment.

NON - INVASIVE METHODS

Radiology

* **X RAY – CHEST & ABDOMEN**

  Radiological changes are found in 60-75% of cases **(Wilmot 1962, Ramachandran et al 1971).** There may be no changes when abscess are small or situated in the lower part of liver.

Radiological signs

1. Elevation of hemi diaphragm.
2. Pleural effusion
3. Pulmonary infiltrates
4. Basal Atelectasis
5. Soft tissue mass may be see in Epigastrium causing Displacement of stomach or colon especially in case of left lobe abscess.

Non Specific Findings

a. Right upper quadrant gas

b. Air – fluid levels within the abscess

c. Ileus

ULTRASONOGRAPHY (USG)

The advent of ultrasonography has opened a new horizon in the diagnosis of liver abscess. It is non – invasive, less expensive, free from radiation hazards, easy and rapid diagnostic procedure with high specificity. Ultrasonogram was done in all cases using 3.5 MHZ sector transducer

* **Advantage - usefulness**

1. Sensitivity of 90-95%
2. Detect lesions as small as 2 cm diameter
3. For detecting the site & number of abscess
4. For guiding the aspiration of abscess
5. For follow up of patients
6. To differentiate from malignant conditions
7. To detect associated Intra abdominal pathology in case of Pyogenic liver abscess

Boultbee and **Ralls** demonstrated the appearance of liver abscess in sonogram. **McCarthy** and **Doust et al** also reported the appearance.

*** Ultrasonographic findings in liver abscess**

1. Cyst homogenicity: Uniform distribution of weak echoes or band like internal echoing, structures representing the walls of loculi or debris

2. Smooth poorly defined wall. In contrast cyst have well defined walls
3. Distal sonic enhancement
4. A location contiguous with liver capsule

* Pyogenic abscess have hypoechoic mass with thick walls with fluid debris level posteriorly. Abscess with bright echogenic foci may contain air or micro bubbles. Necrotic liver tumors also have fluid – filled center and may be confused with abscess but they generally have thicker walls. Nature of Internal echoes correlate to the thickness of the pus. Coarse internal echoes indicate the presence of thick pus and fine internal echoes indicates the presence of relatively fluid pus. Fluid pus noted to be present in early stage of the disease with a short history, while thick pus was present in a patient who has disease for long duration. Fungal abscess will have hypoechoic with echogenic center that are multiple & irregular.

* **Drawbacks**

a. Inaccurate in detecting multiple, small abscess near the dome of diaphragm.
b. Difficult to differentiate liver abscess and tumors with central necrosis
c. Difficult to detect liver abscess in fatty livers.

* After successful therapy complete resolution of ultrasound abnormality may be expected in a period of 6 weeks to 23 months (average 7 months)

C.T. SCAN

Accuracy: 95 – 100%

Indications

1. Procedure of choice for initial assessment of a suspected pyogenic liver abscess.
2. Positive Amoebic serological Test exist but Hepatic Sonogram negative.
3. Detects abscess as small as 0.5 cm. More precise anatomical definition than ultrasound.
4. Better for small abscess near the dome of diaphragm and abscess in fatty livers.
5. For visualizing intra abdominal pathology in case of pyogenic liver abscess such as appendicitis, pancreatic mass, Diverticulitis, Colonic Cancer and Intra peritoneal abscess.

HEPATIC SCINTIGRAPHY

Liver scanning is a valuable aid in the diagnosis and location of liver abscess with sensitivity of 80 – 97%. Commonly used scans are 99 m Technetium, I 131 Rose Bengal or BSP, Indium – 113 scans. They show abscess as non specific hepatic filling defect.

LIMITATIONS

1. Unable to detect lesions smaller than 2 cm
2. Superficial abscess are frequently missed
3. Unable to differentiate between abscess, cyst or tumor
4. Non specific to the etiology of filling defect.

M R I (MAGNETIC RESONANCE IMAGING)

* Detects lesions as small as 0.3 cm

  Superior in defining hepatic venous anatomy

  Useful in patients requiring liver resection to treat the abscess

On M R I. Amoebic abscess often have multiple rims of variable signal intensity. After treatment, the abscess cavity demonstrates concentric rings, Corresponding to an inner area of inflamed tissue with band of collagen and outer margin of inflamed liver tissue.

DIAGNOSTIC LAPAROSCOPY

Laparoscopy offers much more advantage in that you can see an abscess with your naked eye. Although it is an invasive investigation, it is one of the best examinations for visualising an abscess. Not only that but the differential diagnosis of an abscess from conditions like malignancy can be made with elegance. One can also take an open biopsy.

It is worth knowing that superior surface liver abscesses can be better visualised with laparoscopy than at laparotomy. The reason is that at laparoscopy the air insufflation exposes the whole superior surface.

INVASIVE

Cavitogram

Cavitogram were performed by injecting a contrast material viz CONRAY 280 (50ml) into the abscess cavity through the drainage tube (Malecot's or foley's catheter). Most of these cases reveal a collapse of the abscess cavity with resorption.

TREATMENT

Antibiotics

Amoebic abscess

With the introduction of metronidazole in 1960s surgical drainage of most amoebic liver abscess has become unnecessary. Imidazole antibiotics including metronidazole, tinidazole and niridazole will eradicate both intestinal and hepatic amoebic organisms.

A course of metronidazole 750 mg – 3 times a day for 10 days cures approximately 95% of patients with amoebic liver abscess. Most patients shows a prompt therapeutic response to metronidazole with defervescence and decreased abdominal pain within 3-4 days. This response is useful for differentiating amoebic and pyogenic liver abscess in situations where serological testing is unavailable or unpredictable.

* Emetine, dehydroemetine and chloroquine are useful for

    a. Complicated liver abscess
    b. When metronidazole therapy fails.

Dehydroemetine dose – 1-1.5 mg/kg/day…IM-5 days

Tinidazole Dose : 1.2 gm/day – 7 Days.

Because these drugs eliminate invasive organisms (Trophozoites) only simultaneous administration of an intestinal amoebicidal agent for eliminating the cyst, which persist in the intestine even after treatment with metronidazole. Continuation of metronidazole usually fulfills this rule although diiodohydroxyquin, iodoquinol, diloxanide furoate, carbarsone and tetracycline are effective as well. Emetine and dehydroemetine are indicated primarily when patients develop pulmonary complications from amoebic liver abscess.

Chloroquine given in a dose of 600 mg/day for first 2 days them 300 mg/day – up to 21 days. According to **Wilmot A.J & Powell S.J.** addition of chloroquine is valuable because of its specific concentration in the liver.

* Sumeet Bhatia, Dilip R. Karnad, Jyotsna Loak Department of Medicine – King Edmond Memorial Hospital has conducted a Randomised double blind trial of metronidazole VS Secnidazole in Amoebic Liver Abscess and found secnidazole 500 mg tds for 5 days or as a single dose of 1.5 gm/day – for 5 days is as effective as metronidazole in Amoebic Liver Abscess. It is safe and well tolerated.

FOR PYOGENIC ABSCESS

Although the identification of the specific micro organisms involved in pyogenic liver abscess requires appropriate culture information, initial empiric treatment with antibiotics is prudent in most cases. The aetiology of the abscess if known may suggest appropriate antibiotics regimes; however broad spectrum coverage with antibiotics effective against gram – ve rods, streptococcal species and anaerobes should be initiated and continued until definitive organisms are isolated and their sensitivity established. Conventionally a prolonged course of antibiotics, lasting 4-6 weeks is required for patients with a pyogenic abscess. The justification of long term antibiotic therapy has been, the perceived avascularity of both intact and decompressed abscess cavities and the concern for concurrent, but undiagnosed purulent collections within the liver. Prolonged therapy of pyogenic liver abscess with Antibiotics resulted in lower mortality. (VM DAYAL, AK JAIN, JP BHATT, VK DIXIT AK AGARVAL Institute of Medical Science BHU / Varanasi).

Recent studies suggest, however that in adequately drained abscess only 2 weeks of antibiotic administration are necessary. However prolonged antibiotics therapy does remain necessary in patients with multiple or fungal hepatic abscess. In most patients antibiotic treatment must be accompanied by adequate drainage of the abscess cavity. Patients

with multiple, small (< 1.5 cm diameter) abscess, uncomplicated by concurrent surgical disease are best treated with several weeks of antibiotic treatment. Finally most fungal abscess are miliary; therefore they are not amenable to percutaneous or surgical drainage. Prolonged therapy with a systemic antifungal agent is appropriate.

ASPIRATION

* Percutaneous aspiration of abscess with a large bore needle is a safe procedure if done with meticulous aseptic precaution.

INDICATIONS

1. Patients receiving systemic amoebicidal agents yet with symptoms persisting for greater than 72 hours after the drug initiation.
2. To rule out secondary bacterial infection of the amoebic abscess
3. If volume of abscess is greater than 250 ml in volume – to reduce the risk of rupture
4. Those located in left hepatic lobe
5. Lesions associated with marked tenderness and diaphragmatic elevation
6. When metronidazole is contraindicated during pregnancy.

7. In case of pyogenic abscess aspiration provides valuable diagnostic and bacteriologic data and for young patients and if there is not co-existing intra abdominal pathology.
8. Sero Negative abscess

   With ultra sonogram and CT scan localization & aspiration is easier and safe. The needle should not be introduced deeper than 10 cm. as portal vein may be entered.

Three usual complications are hemorrhage, tear of the liver and secondary infection.

Aspiration is **contraindicated** in doubtful cases, suspected cases of hydatid cysts and in patients with bleeding tendency and also in hepatic malignancy. Balasegram described the complications of leakage which may follow needle aspiration namely subphrenic abscess (14%) and peritonitis (9%)

PERCUTANEOUS DRAINAGE

Recommended for selected cases of abscess.

Indications

1. Thick viscous contents of an abscess
2. Failure of repeated aspiration

3. Large abscess bulging through the abdominal wall.
4. Most useful for the management of pulmonary, peritoneal and pericardial complications.

* Average length of catheter drainage lasting from 11-19 days. Inadequate drainage and subsequent treatment failure most commonly results from catheter obstruction with purulent (obstruction) material or untreated loculations within the abscess. Percutaneous drainage is also frequently unsuccessful in immuno compromised patients.

Contraindications

1. Associated biliary or Intra abdominal pathology requiring simultaneous operative intervention.
2. Coagulopathy and anatomical inaccessibility preclude percutaneous catheter placement, owing to the increased risk of life threatening hemorrhage or violation of hollow viscus.
3. Multiple abscess and generalized ascites are relative contraindications.

Complications

Sepsis, Hemorrhage, contamination of the pleural & peritoneal cavity. Bowel perforation, Catheter related pain, Catheter displacement.

SURGICAL DRAINAGE

Surgical drainage procedures now are performed less frequently than in the past khanna et al recommended open drainage for

Indications

1. Most common indication is failure of conservative therapy
2. When amoebic liver abscess erodes into neighboring viscus
3. Patients wit septicemia from secondarily infected amoebic abscess
4. In case of pyogenic abscess laparotomy is indicated for patients with co-existing biliary pathology, such as biliary stone or strictures
5. Patients with loculated or multiple abscess, abscess inaccessible to percutaneous drainage or involving entire lobe.
6. Shock with multi system organ failure.
7. Respiratory and Renal failure.
8. Weight loss greater than 10 Kgs.
9. Albumin less than 3 gm/ dl.

Approaches:

a. Transperitoneal approach

Transperitoneal approach allows for abscess and abdominal exploration to identify previously undetected abscess and location of an etiological source.

b. Posterior Transpleural approach

For high Posterior lesions, a posterior Transpleural approach is performed. Although this allows easy access to the abscess, the identification of multiple lesions or a concurrent abdominal pathology is not possible.

***** Transperitoneal operative drainage is now the standard treatment for most patients.

HEPATIC RESECTION

* Rarely required

Indications

1. Isolated lobar involvement with single or multiple non healing abscess.
2. Patients with infected hepatic malignancies, Hemobilia & chronicgranulomatous disease.

MANAGEMENT OF COMPLICATED LIVER ABSCESS

Management of pleuropulmonary complication

Amoebic liver abscess rupturing into pleural cavity is usually managed conservatively with Intercostal drainage. Most of the patients, resolve with this treatment. If this is not drained adequately then we have to do decortication. In case of Broncho Pleural fistula the treatment is also conservative. ICD is enough. If there is residual abscess in the bronchous not getting resolved with ICD then we have to resect the particular segment of lung. If there is broncho Pleuro Biliary fistula then we have to resect the diseased bronchous and suture the Biliary fistula.

Management of peritoneal complications

Acute generalized peritonitis with shock due to sudden rupture of an Amoebic Liver Abscess requires surgical treatment. Conservative management of intraperitoneal requires of Amoebic liver abscess was associated with a mortality of 75% to 100%. Laparotomy with complete toileting of the liver abscess as well as the peritoneal cavity have shown recoveries of 50 percent to 100 percent.

Management of pericardial complications

Pericardial drainage will be enough for abscess rupturing into the pericardial cavity but if is not doing well and the patient is not improving well then we have to do pericardiectomy. The prognosis is not good.

PROGNOSIS

* **POOR**

1. Age > 70 Years
2. Diabetes mellitus
3. Associated malignancy
4. Multiple abscess
5. Septicemia
6. WBC > 20,000
7. Biliary etiology
8. Poly microbial bacterimia
9. Increased Bilirubin
10. Albumin Level < 2 gm / dl
11. Pulmonary Complications
12. Rupture
13. Late presentation
14. Increased SGOT
15. Aerobic abscess

MATERIALS AND METHODS

The material used in this study consisted of 110 cases of liver abscess which were admitted in the Department of General Surgery Government Kilpauk Hospital Chennai – 10 during the year 2003 – 2006.

CRITERIA FOR INCLUSION

1. Enlarged and tender liver.
2. Presence of Macroscopic and Microscopic features of pus in the liver.
3. Culture and sensitivity of aspirated pus.
4. Radiological evidence of raised and fixed right dome of diaphragm.
5. Ultrasonogram evidence of liver abscess.

* The pathophysiology, Clinical behaviour and investigative modalities and treatment patterns are thoroughly analyzed and presented here.

RESULTS OF STUDY

TABLE

Age distribution in Liver Abscess

Age in Years	No. of Cases	Percentage
10 – 20	0	0
21 – 30	15	14
31 – 40	25	23
41 – 50	31	28
> 50	39	35

Sex Distribution in Liver Abscess

Sex	No. of Cases	Percentage
Male	96	87
Female	14	13

CLINICAL PRESENTATION

Clinical Features	No. of Cases	Percentage
1. Abdominal Pain	105	95
2. Intercostal Tenderness	43	39
3. Enlarged Liver	48	44
4. Fever	71	65
5. H/o. Dysentery	6	5
6. Nausea and Vomiting	28	25
7. Anorexia	39	35
8. Loss of Weight	22	20
9. Jaundice	15	14
10. Hiccup	2	2
11. H/o. Alcohol	90	82

MODES OF PRESENTATION

Presentation	No. of Cases	Percentage
1. Classical	85	77
2. Peritonitis	15	14
3. Rupture into Thoracic Cavity	4	4
4. Silent	6	5

LABORATORY INVESTIGATIONS

Hematological investigation

Investigation	Value	No. of Cases	Percentage
Haemoglobin	7 – 10 gms. / dl.	74	67
	10 – 14 gms. / dl	36	33
WBC Count	5,000 – 10,000 cells / cu.mm.	46	42
	10,000 – 15,000 cells / cu.mm.	56	51
	> 15,000 cells / cu.mm.	8	7
ESR	< 15 mm > 15 mm	11 99	10 90

LIVER FUNCTION TESTS

Investigation	No. of Cases	Percentage
1. Increased Bilirubin	58	53
2. Increased Serum Alkaline Phosphatase	61	55
3. Decreased Serum Albumin	22	21

RADIOLOGICAL INVESTIGATION

X-ray Chest and X-ray abdomen

Radiological Feature	No. of Cases	Percentage
Raised dome of Diaphragm	52	47
Pleural effusion	15	14
Basal atelectasis	11	10
Enlarged Liver	40	36
Ground Glass appearance	7	6

ULTRASONOGRAM

In our present study all cases were analysed with 2 dimension ultra sonography.

USG DATA – REGARDING SIZE

Size in Cms.	No. of Cases	Percentage
< 5	21	19
5 – 10	60	55
> 10	29	26

DISTRIBUTION OF ABSCESS

Right Lobe		Left Lobe		Both Lobes
78 (70%)		16 (15%)		16 (15%)
Single	**Multiple**	**Single**	**Multiple**	
56 (51%)	22 (20%)	16 (15%)	0	

Right Lobe is involved predominantly (70%)

MODES OF TREATMENT

Mode of Treatment	No. of Cases	Percentage
Conservative	20	18
Aspiration	78	71
Percutaneous catheter drainage	5	5
Open Drainage	7	6

BACTERIOLOGICAL STUDY

No. of persons found to have bacterial growth	Organisms isolated
6 (5%)	E. coli Klebsiella Pseudomonas

TROPHOZOITE AND CYST OF E.HISTOLYTICA RECOVERED

Stools	Aspirated Pus
16 (15%)	10 (9%)

QUANTITY OF PUS ASPIRATED

Quantity	No. of Cases	Percentage
< 100 ml	22	20
100 - 500 ml	39	35
500 – 1000 ml	5	5
> 1000 ml	12	11

DISCUSSION

The condition of hepatic abscess and its grave prognosis were known in ancient times to **Hippocrates** (460 BC – 370 BC) and **Celsus** (53 BC – 7 AD) Hippocrates was able to distinguish from cystic liver disease. Celsus appreciated the poor prognosis of hepatic abscess associated with Jaundice. Not until 1936 did **Bright** in his own observation on Jaundice clearly described hepatic suppuration with true abscess formation.

Oschner, Debakey and Murray 1938 in their classis article reported amoebic liver abscess, 75% as very common in the warmer southern climate. Liver abscess though a well defined clinical entity yet many difficulties were faced in determining the site, size and number of abscess. **Chuttani et al** 1963 have commented that the difficulties in clinical diagnosis of hepatic amoebiasis can be diverse and real. Those who do not meet the condition frequently are not likely to appreciate them fully. Untreated liver abscess has mortality rates approaching 100%. Reports of successful medical management with or without aspiration describe case fatality rates as low as 6%.

In our study out of 13,500 cases admitted in our hospital in 2003 to 2006. We are reported about 110 cases of liver abscess. Incidence being

0.8%. **In our study** peak age incidence was noticed in 5th decade followed by Fourth and Sixth decade. In our study there is more number of cases in low socio economic status. According to **Garewal** the highest incidence was noted in people who consume alcohol and also in people who live with poor hygienic conditions, contaminated drinking water, malnutrition, hepatic dysfunction and low host resistance.

Highest incidence of liver abscess in males 96% in our study has been attributed to alcoholism, (Present study H/o alcoholism was present in 82% Cases). This correlates with the study of **Oschner & Debakey** which predispose to hepatitis. Alcohol produces heptaocellular damage and may make it prone to develop hepatic abscess – **Sheila – Sherlock.**

Presentation	**Present Study (n=110) %**	**D.S Sing et al (1980) (n=42) %**	**Barnas et al (1987) n=96 %**
Pain Right Hypochondrium	95	100	67
Fever	65	100	87
H/o. Diarrhoea/ Dysentry	5	85	35
Jaundice	14	24	10
Weight Loss	20	-	10
Appetite	35	-	45
Breathelessness	5	-	24

In our study commonest symptom being abdominal pain and fever. Commonest sign being Intercostal Tenderness and Tender hepatomegaly.

In present study, anemia was noted in 67% cases and Jaundice in 14% patients especially in Pyogenic liver abscess patients.

COMPARATIVE STUDY

Test	Ramachandran % (1976)	Sharma % (1980)	Prasad % (1987)	Present % (2003)
Hyper Bilirubinemia	8	10	7	53
Hypoalbuminemia	42	64	57	55
Increased Serum Alkaline Phosphatase	50	37	41	21

Data concluded in his study that Jaundice in liver abscess is primarily of cholestatic origin. Intrahepatic cholestasis which is due to compression of Both hepatic ducts. Though **Lamot and Pooler, Vakil et al, Hazra et al have noted an increase mortality** in liver abscess with Jaundice we have not encounted such thing in this study.

NON INVASIVE METHODS

PLAIN X RAY CHEST & ABDOMEN

In the present study raised right hemidiaphragm was noted in 47% cases and enlarged liver in 36% of cases.

ULTRASONOGRAPHY

The advent of Ultrasonography has opened a new horizon of the diagnosis of hepato biliary disease. Sonography is of immense help in finding the abscess cavity, its location, number and valuable in aspiration and follow up. It is also useful in finding out the nature of PUS, So that helpful in determining the size of needle for aspiration.

In our study the sensitivity of ultrasonogram is around 97% However false positive results (3%) was encounted in this study turned out to be degenerating Hepatomas.

Similarly **Cimmino CV. Scott DW** reviewed as case report of a benign liver tumor with central necrosis which was misdiagnosed clinically as liver abscess.

COMPARATIVE STUDY

LOBE INVOLVEMENT – USG

Author	Right %	Left %	Both %
RALLS (1979)	83.3	16.7	-
BOULT BEE (1979)	77	15	8
PRESENT (2003)	70	15	15

NUMBER OF ABSCESS CAVITY

Number	Present Study %	K.L. Ghose et al %
Single	65	86
Multiple	35	13

LEFT LOBE ABSCESS

Study Groups	Left Lobe Abscess %
De Bakey and Ochsner	10
Abdul Khair et al	36
Harinasuta et al	8
Wilmot	13
Present Study	15

All right Lobe Abscess were diagnosed by clinical enlargement of liver whereas such definite Lobe enlargement was not seen in Left Lobe Abscess.

The highest incidence of right lobe abscess in this study (70%) correlated with previous study.

MANAGEMENT

In the present study the diagnosis of liver abscess mainly depends upon the clinical feature and ultrasonography and to lesser extent with stool and pus examination.

*** CONSERVATIVE TREATMENT**

In the present study we had a **protocol** of managing the liver abscess of size **less than 5 cm on ultrasonogram with conservative management (drugs).**

We used to treat the amoebic liver abscess patients with Ciprofloxaxin 200 mg twice daily, metronidazole 500 mg thrice daily both parenterally for five days (and then changed to oral preparation) along with chloroquine 300 mg twice daily orally.

Pyogenic liver abscess were treated first with empirical antibiotics – Ampicillin Gentamycin and Metronidazle and then changed according to culture and sensitivity we haven't faced any major complications of chloroquine except vomiting. Most of the patients (90%) resolve and do better with conservative management. About 10% patients whose size doesn't decrease with antibiotics even after 4-5 days were aspirated under ultrasound guidance.

* ASPIRATION

Out of 110 cases studied, for about **78 cases** closed needle ultrasound guided aspiration was done. We did aspiration using 14 gauge needle for the patients in whom conservative line of management fails and for abscess of more than 5 cm size. Prior to Aspiration we routinely did bleeding time and clotting time. Injection vitamin K. one ampule was given daily for 3 days prior to aspiration. When anti amoebicidal a drug has been given, thick pus begins to liquefy and it can be aspirated 3 days later under ultrasonogram control.

No biliary peritonitis was encounted in this study, where as **Balasegaram** has described the complications of leakage which may follow needle aspiration eg. Subphrenic abscess (14%) and peritonitis (9%). No such complication arise in our study.

Generally we treated the patients with parenteral antibiotics for 5 days and then changed to oral antibiotics. After discharging the patients we advised them to continue tablet metronidazole for 3 weeks and T.Chloroquine for 2 weeks. We reviewed the size of the abscess with ultrasonogram before discharge and then every 2 months, until the abscess resolves. In our present study it took 3-4 months for complete resolution of abscess by ultrasonogram.

* PERCUTANEOUS DRAINAGE

We did percutaneous drainage for 5 cases. Among 5 cases of percutaneous drainage, we did ICD for 4 cases. Out of 4 cases 2 cases expired. 1 case expired on 4th POD and another expired on 2nd POD, amounting to 50% mortality in our study.

* LAPAROTOMY

With the advent of modern imaging techniques the role of open drainage of hepatic abscess is almost negligible.

We did laparotomy and open drainage for 7 cases which were presented as

a. Acute abdomen where abscess burst presenting as perforated peptic ulcer, pancreatitis, ileal perforation or as perforated appendicitis.
b. Patients not responding to aspiration.
c. For very large abscess more than 10 cm.
d. For large multiple abscess.

Open drainage done with malecot's or foley's catheter in situ. All patients did well.

SUMMARY AND CONCLUSION

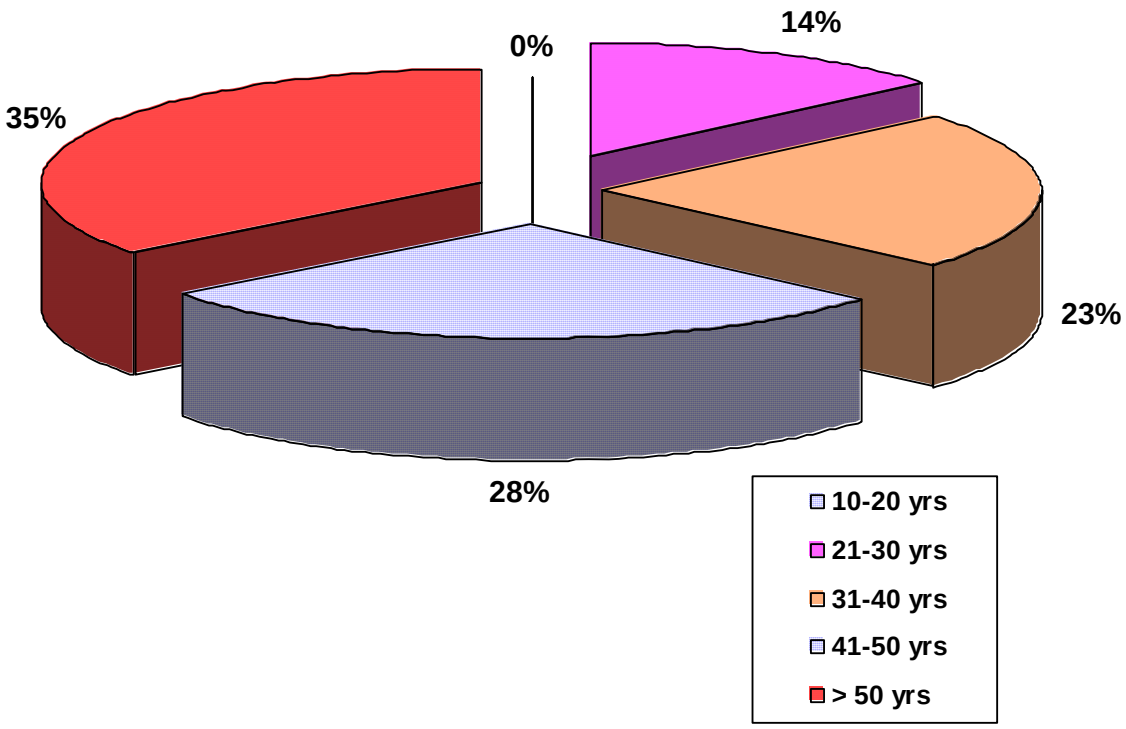
Out of 110 cases taken out for study majority presented with classical features.

1. Incidence of liver abscess is **0.8%** of total admissions in our hospital. Incidence of Amoebic liver abscess is very common in our study. The ratio of amoebic liver abscess: pyogenic liver abscess being 19:1
2. About 82% of patient were alcoholic.
3. Male predominates both in amoebic and pyogenic liver abscess in the ratio of 7:1.
4. Anemias, Leucocytosis were common Accompaniments.
5. Commonest symptom is abdominal pain and fever, sign being Tender hepatomegaly and Intercostal Tenderness.
6. Only 14% of patients presented with Jaundice
7. Right Lobe was predominantly involved in the ratio of 5:1
8. Clinical diagnosis of liver abscess is straight forward except for those presenting with complications.
9. Only 15% of patients, we are able to isolate E. Histolytic in stools & 9% from PUS.

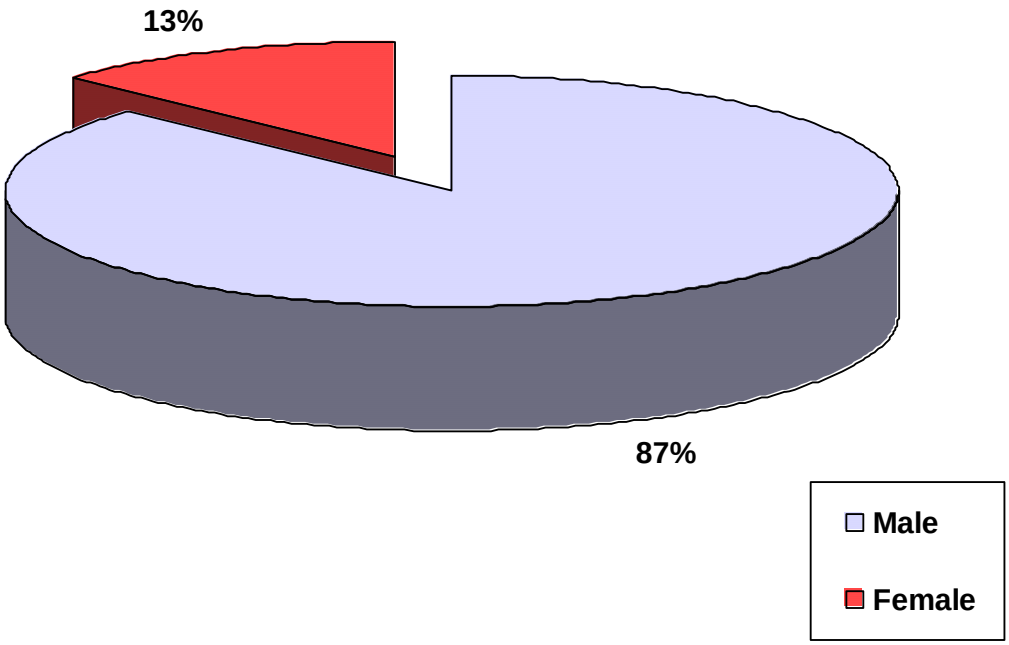
10. Out of 110 cases treated, 20 cases were treated conservatively, 78 cases required aspiration, 5 cases required percutaneous catheter drainage and 7 cases required open drainage.
11. Ultrasound is the commonest and most useful investigation for diagnosis, treatment as well as follow up.
12. CT Scan mainly reserved for doubtful cases and those presenting with complications.
13. In our series, there is more number of abscess seen in low socioeconomic status patients. 30% of patients are in High Socio Economic status, 70% of patients are in low Socio Economic Status.
14. Mortality in our series

Uncomplicated liver abscess	:	No mortality
Ruptured into abdominal cavity	:	No mortality
Ruptured into thoracic cavity	:	50% Mortality

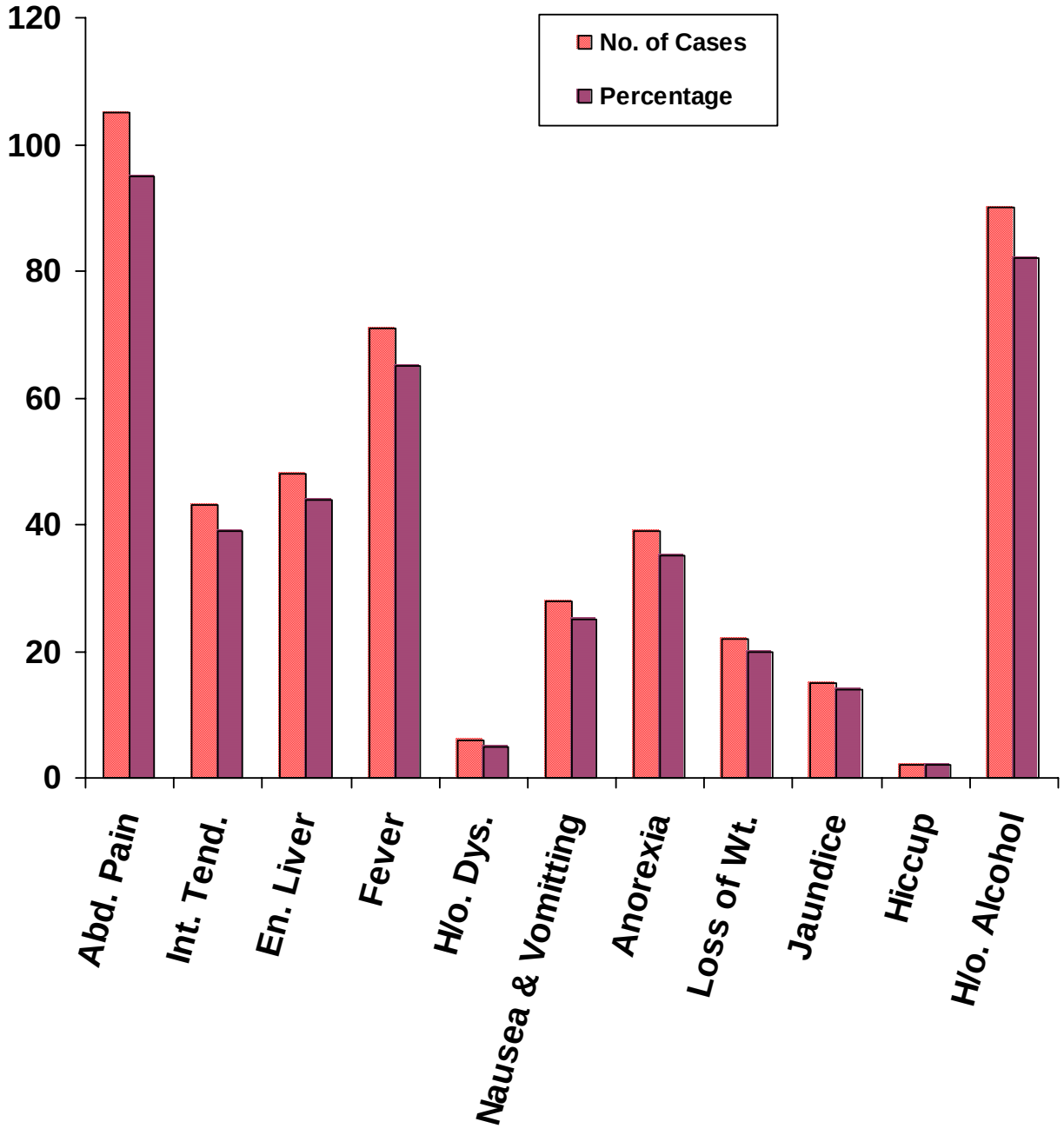
AGE DISTRIBUTION IN LIVER ABSCESS



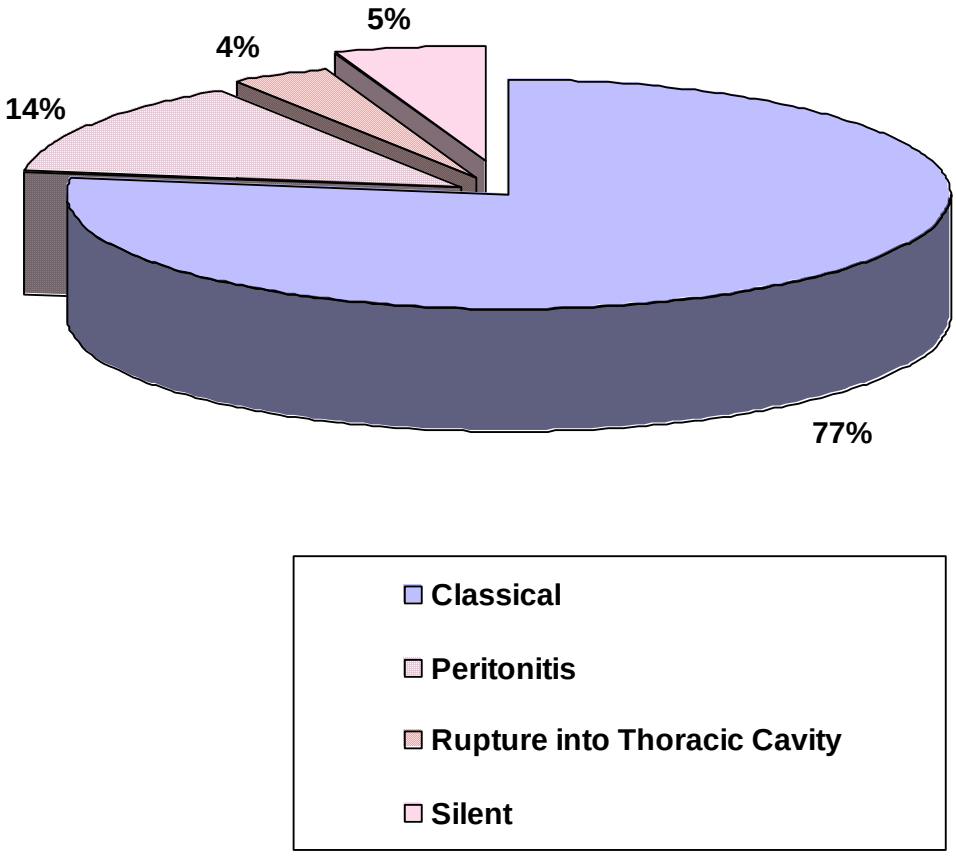
SEX DISTRIBUTION IN LIVER ABSCESS



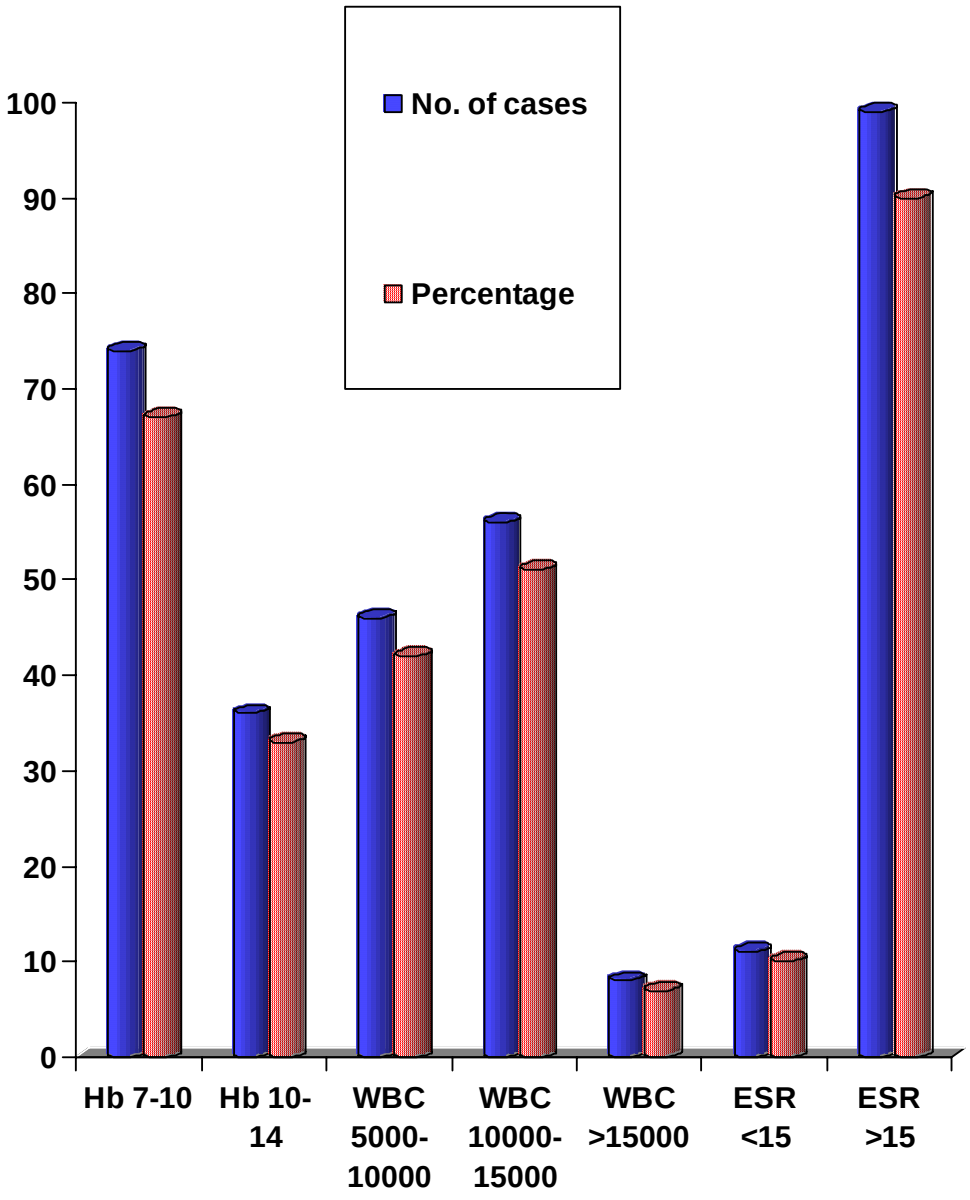
CLINICAL PRESENTATION



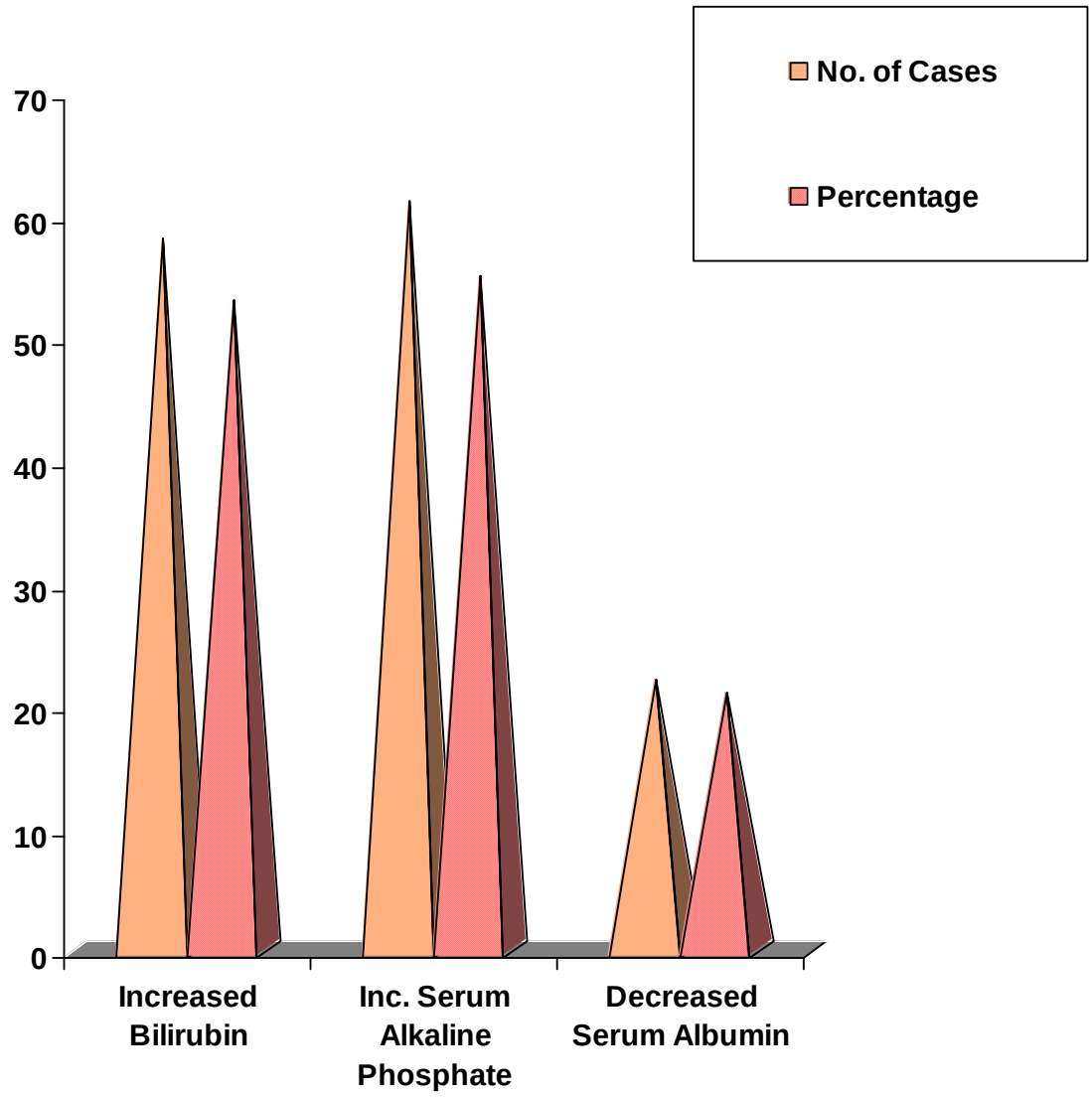
MODES OF PRESENTATION



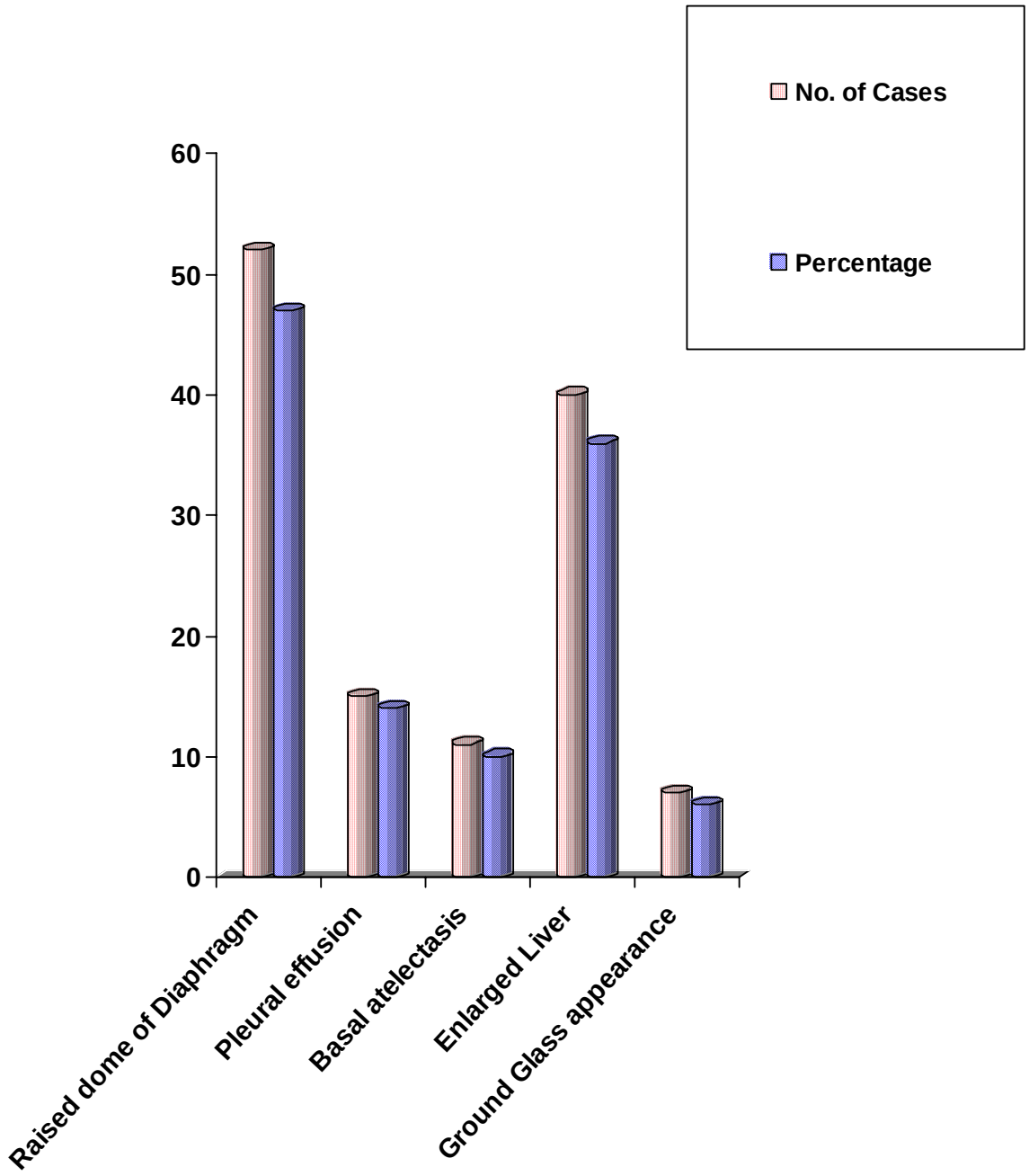
HEMATOLOGICAL INVESTIGATION



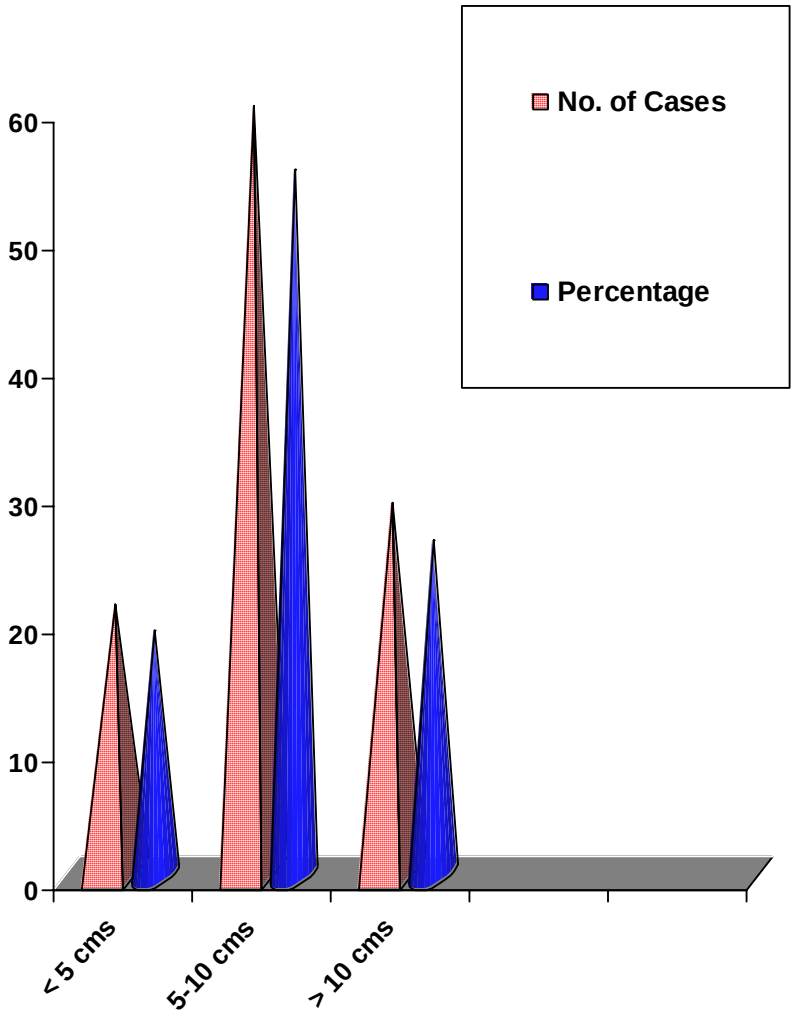
LIVER FUNCTION TESTS



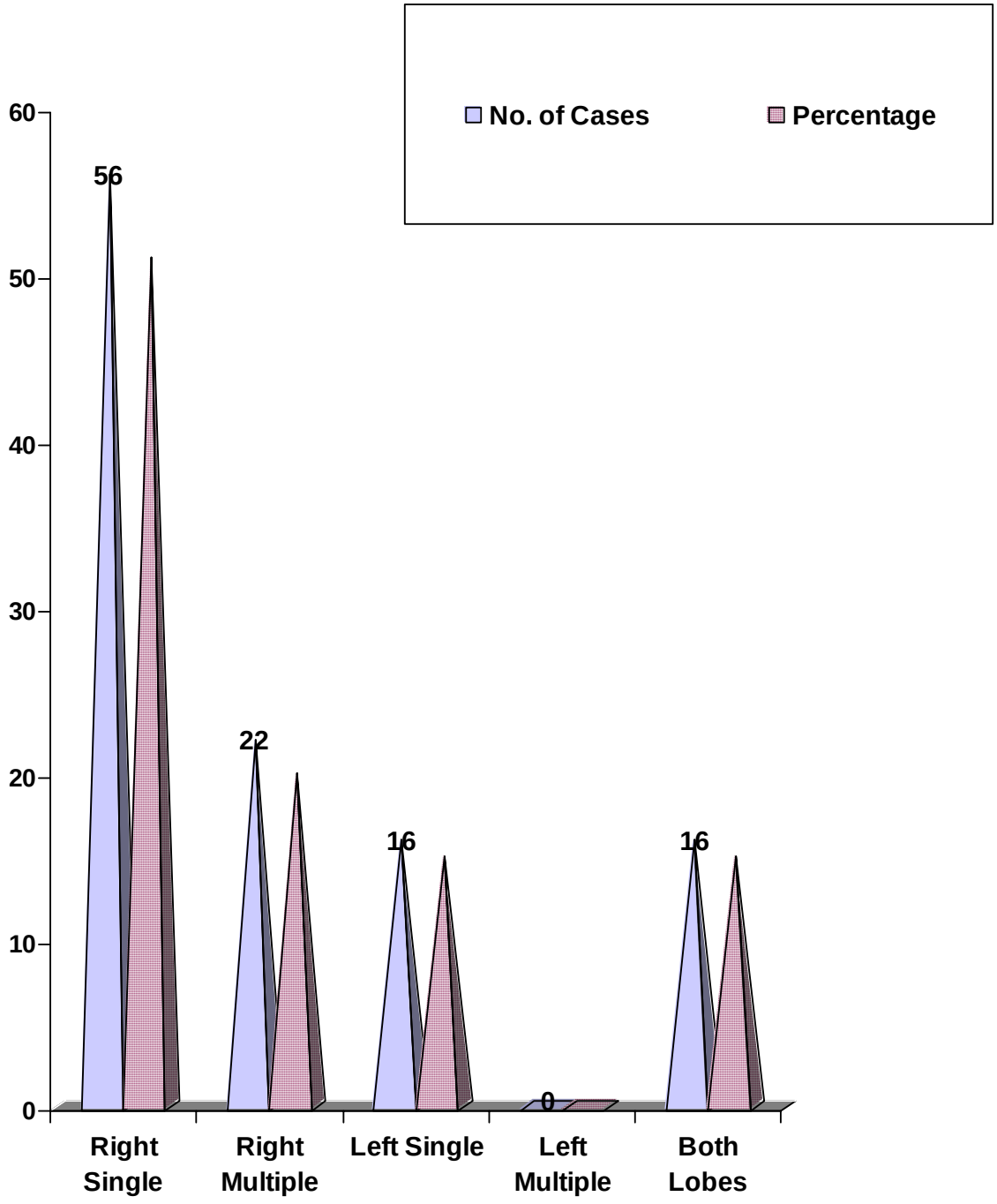
X-RAY CHEST AND X-RAY ABDOMEN



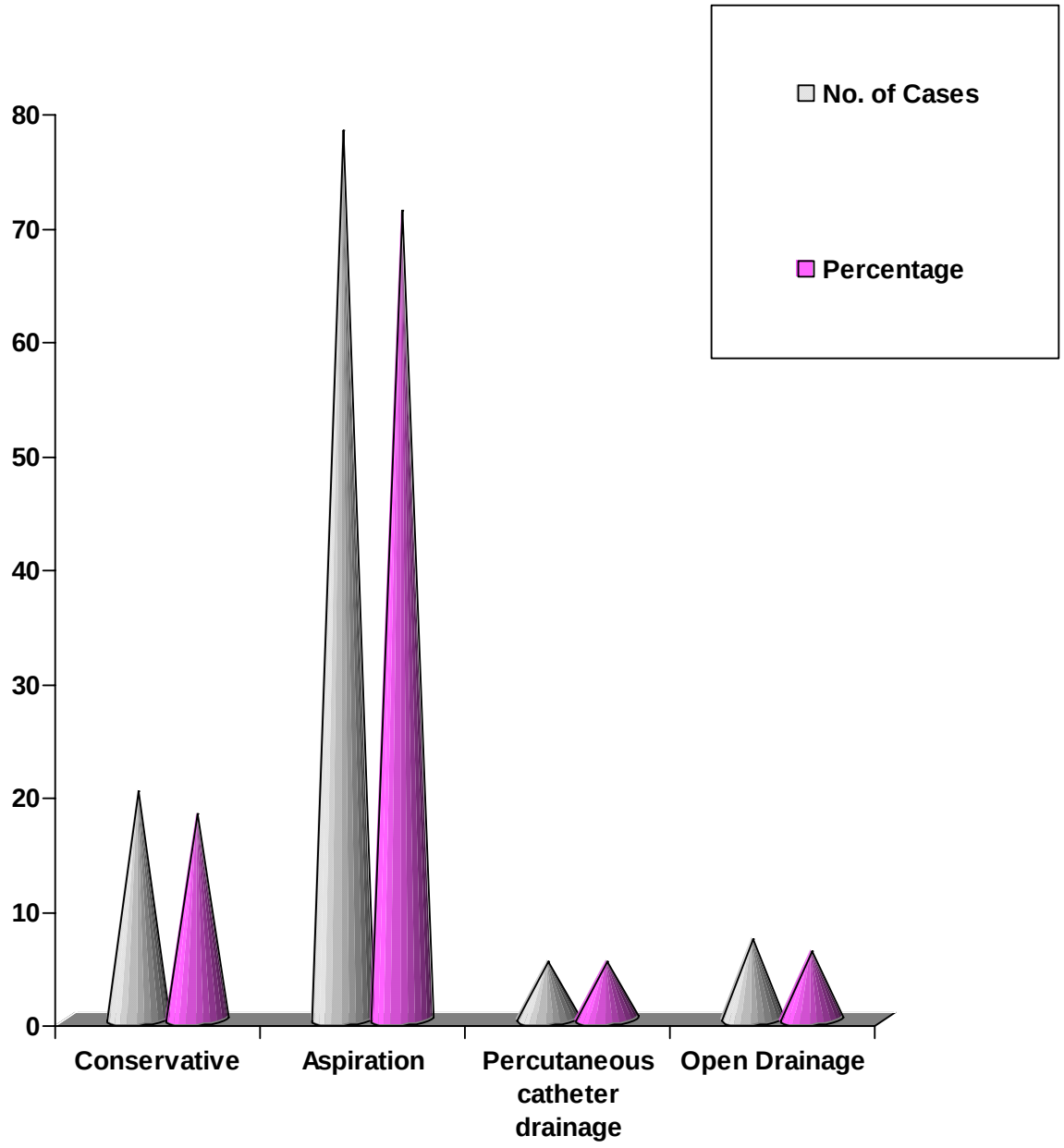
USG DATA – REGARDING SIZE



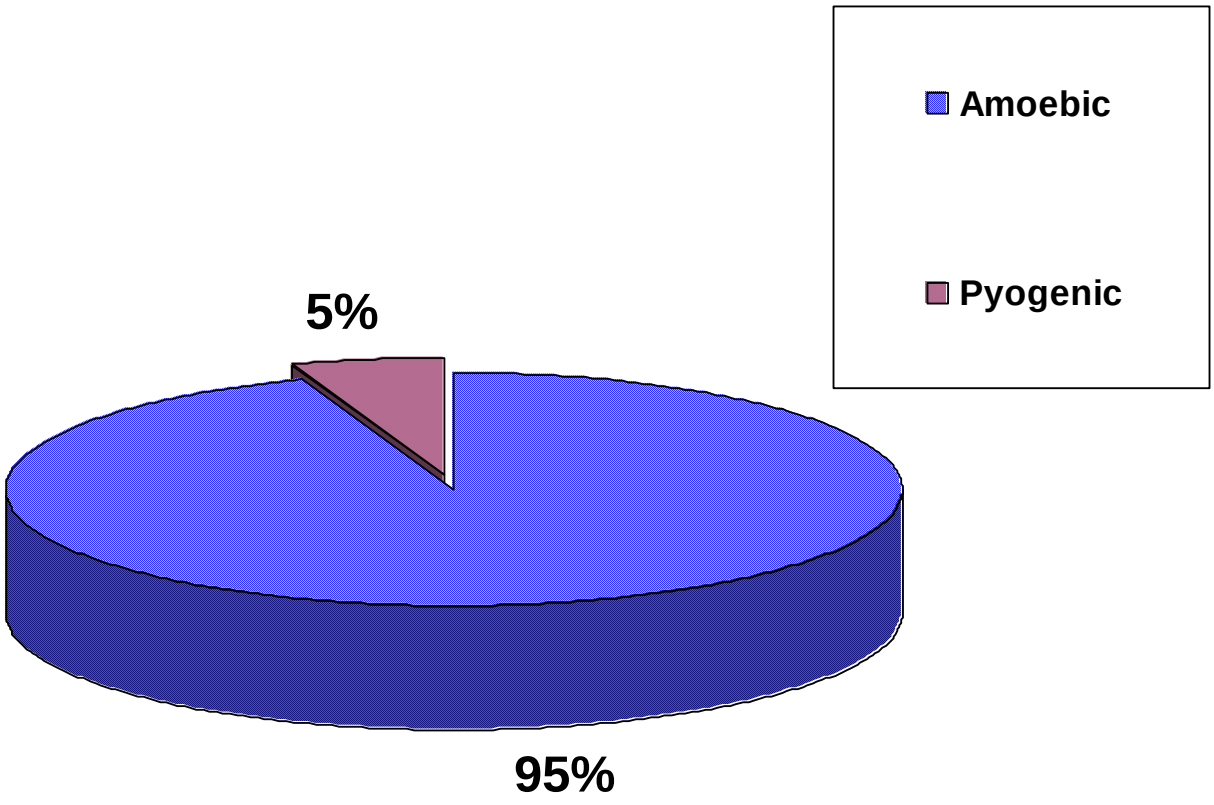
DISTRIBUTION OF ABSCESS



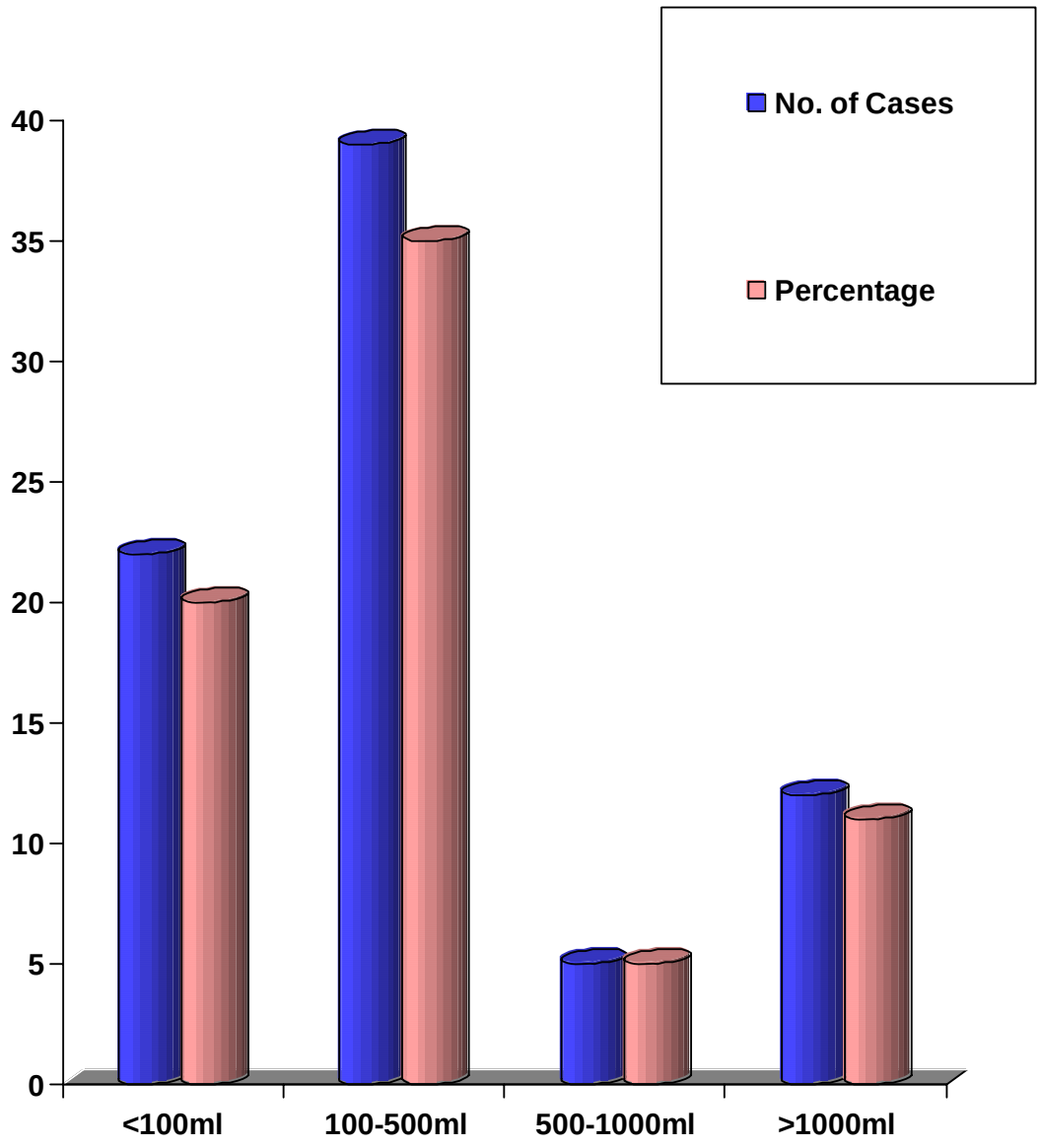
MODES OF TREATMENT



BACTERIOLOGICAL STUDY



QUANTITY OF PUS ASPIRATED



MASTER CHART

S. No.	Name	Age / Sex	IP No.	Treatment
1.	Vadivel	70/M	15498	Aspiration
2.	Subramaniam	60/M	16880	Aspiration
3.	Mahendran	44/M	8034	Aspiration
4.	Gajendran	28/M	12169	Conservative
5.	Balasubramani	58/M	12664	Conservative
6.	Balu	47/M	9624	ICD & Aspiration
7.	Kannan	23/M	13868	Percutaneous Drainage
8.	Ravichandran	33/M	14079	Aspiration
9.	Thangavel	60/M	15019	Aspiration
10.	Kathija	20/F	16264	Conservative
11.	Ramesh	28/M	19110	Aspiration
12.	Baskar	30/M	19373	Conservative
13.	Jayaraj	30/M	19558	Aspiration
14.	Shanmugham	22/M	20143	Aspiration
15.	Velu	23/M	21805	Aspiration

S. No.	Name	Age / Sex	IP No.	Treatment
16.	Perumal	26/M	22265	Aspiration
17.	Velu	31/M	23271	Aspiration
18.	Shanmugasundaram	63/M	949	Laparotomy & Open drainage
19.	Babu	50/M	3136	Percutenous drainage
20.	Kanniappan	42/M	4757	Aspiration
21.	Sunderaj	23/M	6226	Aspiration
22.	Perumal	58/M	7745	Laparotomy & Open drainage
23.	Shanmugham	47/M	9076	Aspiration
24.	Rajendran	53/M	10258	Conservative
25.	Koteeswaran	47/M	14026	Aspiration
26.	Gunasekar	38/M	14608	Laparotomy & Open drainage
27.	Kalimuthu	35/M	20331	Aspiration
28.	Gunasekar	38/M	16797	Aspiration
29.	Gunasekaran	47/M	19477	Aspiration
30.	Mohan	35/M	20230	Aspiration

S. No.	Name	Age / Sex	IP No.	Treatment
31.	Devaraj	55/M	21675	Aspiration
32.	Rajendran	38/M	21692	Aspiration
33.	Pandu	38/M	22061	Conservative
34.	Manibalan	48/M	24228	Aspiration
35.	Thangavelu	42/M	24415	ICD & Aspiration
36.	Doraiswamy	60/M	26493	Laparotomy & Open drainage
37.	Nagaraj	55/M	35903	Aspiration
38.	Perumal	45/M	36158	Aspiration
39.	Moorthy	37/M	156	Percutaneous drainage
40.	Kuppuswamy	85/M	9939	Aspiration
41.	Devadoss	40/M	10558	Aspiration
42.	Kannan	28/M	11862	Aspiration
43.	Gunasekar	38/M	14608	Aspiration
44.	Chandran	45/M	15005	ICD & Aspiration
45.	Deenadayalan	52/M	16530	Aspiration

S. No.	Name	Age / Sex	IP No.	Treatment
46.	Pandi	40/M	16110	Aspiration
47.	Krishnan	55/M	22796	Aspiration
48.	Venkatesh Kumar	35/M	15988	Aspiration
49.	Ramanathan	41/M	17303	Conservative
50.	Perumal	45/M	17927	Aspiration
51.	Babu	23/M	19737	Aspiration
52.	Kalimuthu	35/M	20331	Aspiration
53.	Sakunthala	50/F	20708	Aspiration
54.	Devaraj	55/M	21675	Aspiration
55.	Palani	49/M	21318	Aspiration
56.	Rajendran	38/M	21692	Aspiration
57.	Govindaraj	55/M	24081	Conservative
58.	Mohan	35/M	20230	Laparotomy & Open drainage
59.	Subramani	45/M	23946	Aspiration
60.	Thangavelu	42/M	24415	Aspiration

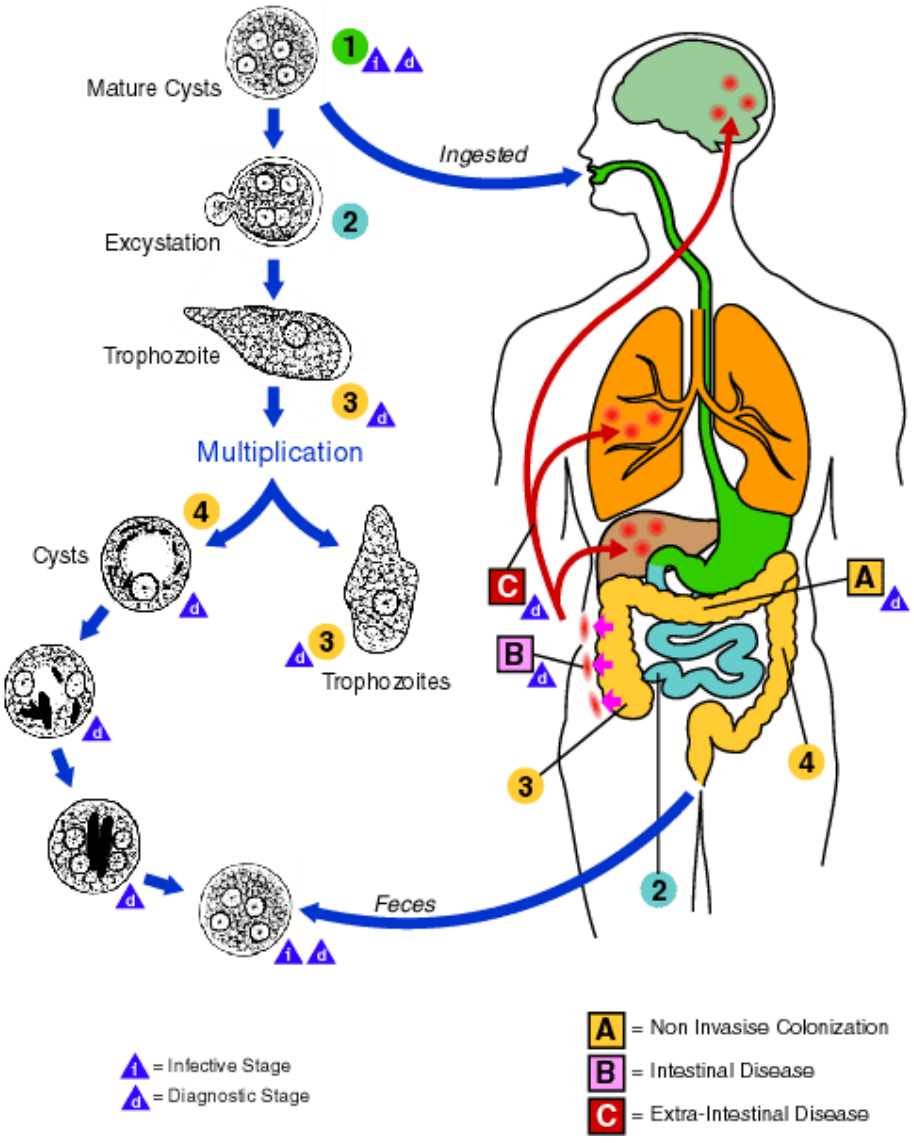
S. No.	Name	Age / Sex	IP No.	Treatment
61.	Mani	55/M	24077	Percutaneous Drainage
62.	Nithyanandam	55/M	23047	ICD & Aspiration
63.	Sakunthala	50/M	23024	Aspiration
64.	Balu	53/M	25418	Percutenous Drainage
65.	Zerina	22/F	26224	Aspiration
66.	Vellai Pillai	55/M	27731	Aspiration
67.	Shanthi	38/F	29688	Aspiration
68.	Murugan	60/M	31381	Aspiration
69.	Sukumar	40/M	31413	Conservative
70.	Chinnaswami	60/M	33287	Laparotomy & Open drainage
71.	Nandhini	27/F	34274	Aspiration
72.	Rani	61/F	35127	Conservative
73.	Nagaraj	55/M	35903	Aspiration
74.	Perumal	45/M	38158	Conservative
75.	Ranganathan	65/M	332	Aspiration

S. No.	Name	Age / Sex	IP No.	Treatment
76.	Venkatesan	29/M	1270	Aspiration
77.	Divyanandan	33/M	1631	Aspiration
78.	Moorthy	37/M	156	Aspiration
79.	Sargunaraj	28/M	1356	Aspiration
80.	Kamala	65/F	1403	Conservative
81.	Mohan	35/M	34094	Aspiration
82.	Shantha	45/F	2879	Aspiration
83.	Sukur	55/M	3932	Conservative
84.	Arumugam	45/M	2925	Aspiration
85.	Shantha	45/F	4560	Aspiration
86.	Jayakumar	35/M	5405	Aspiration
87.	Sakunthala	40/F	6165	Aspiration
88.	Arasu	39/M	8205	Conservative
89.	Mani	46/M	7802	Conservative
90.	Latha	30/F	11809	Aspiration

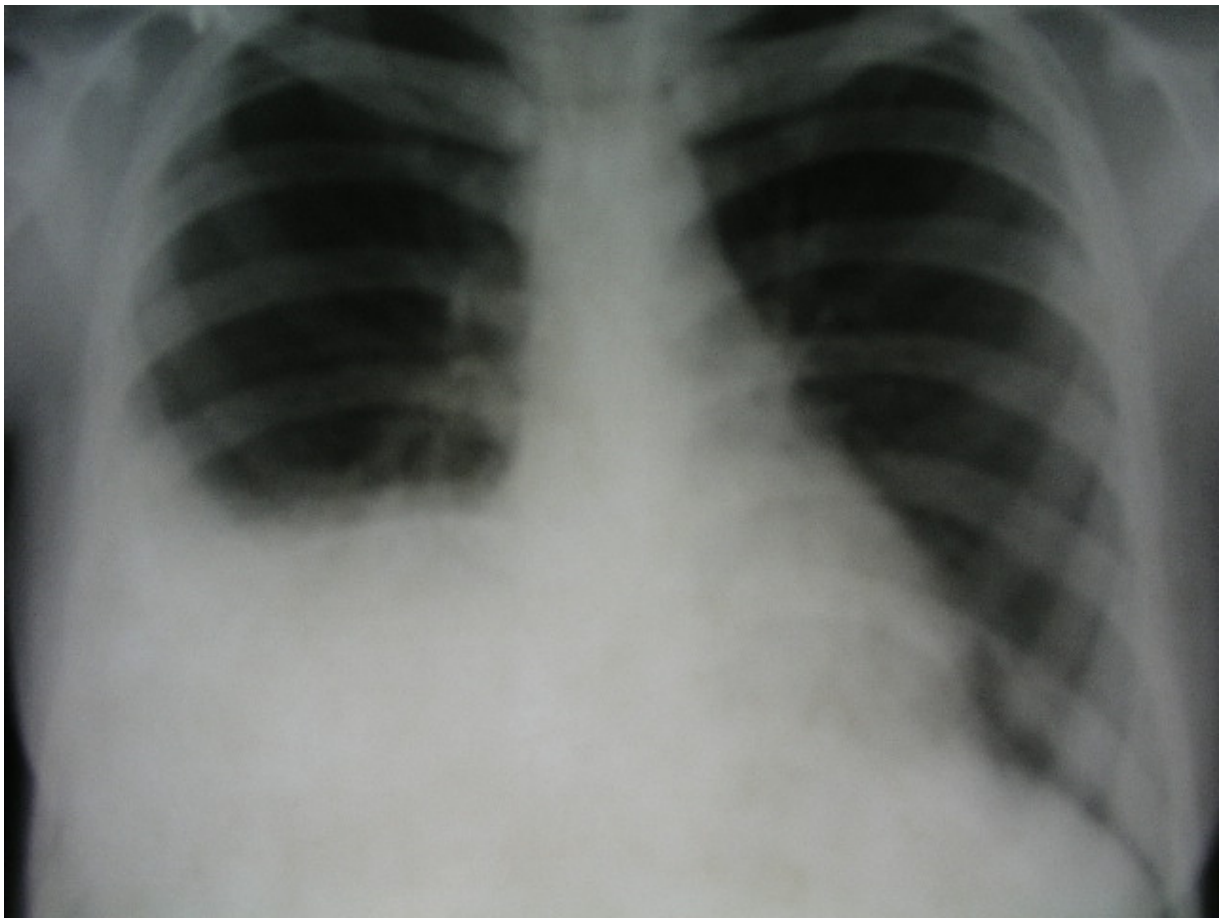
S. No.	Name	Age / Sex	IP No.	Treatment
91.	Shanmugham	35/M	11561	Conservative
92.	Karunanidhi	34/M	12270	Aspiration
93.	Chockalingam	58/M	12035	Aspiration
94.	Kanniappan	48/M	13552	Aspiration
95.	Karunanidhi	54/M	13007	Aspiration
96.	Sai Baba	46/M	14160	Conservative
97.	Vadivel	70/M	15490	Aspiration
98.	Kanniappan	48/M	16242	Aspiration
99.	Subramanian	60/M	16550	Conservative
100.	Dhanam	50/F	16741	Aspiration
101.	Duraiswami Pillai	80/M	16925	Conservative
102.	Kannan	50/M	16368	Aspiration
103.	Soundarapandian	53/M	16936	Aspiration
104.	Mahendran	34/M	18929	Aspiration
105.	Subramaniam	55/M	19613	Aspiration

S. No.	Name	Age / Sex	IP No.	Treatment
106.	Uthirapathy	65/M	20031	Conservative
107.	Ramaswamy	55/M	17834	Aspiration
108.	Ellammal	52/F	22490	Aspiration
109.	Sakunthala	48/F	12184	Laparotomy & Open drainage
110.	Ganesan	51/M	22929	Aspiration

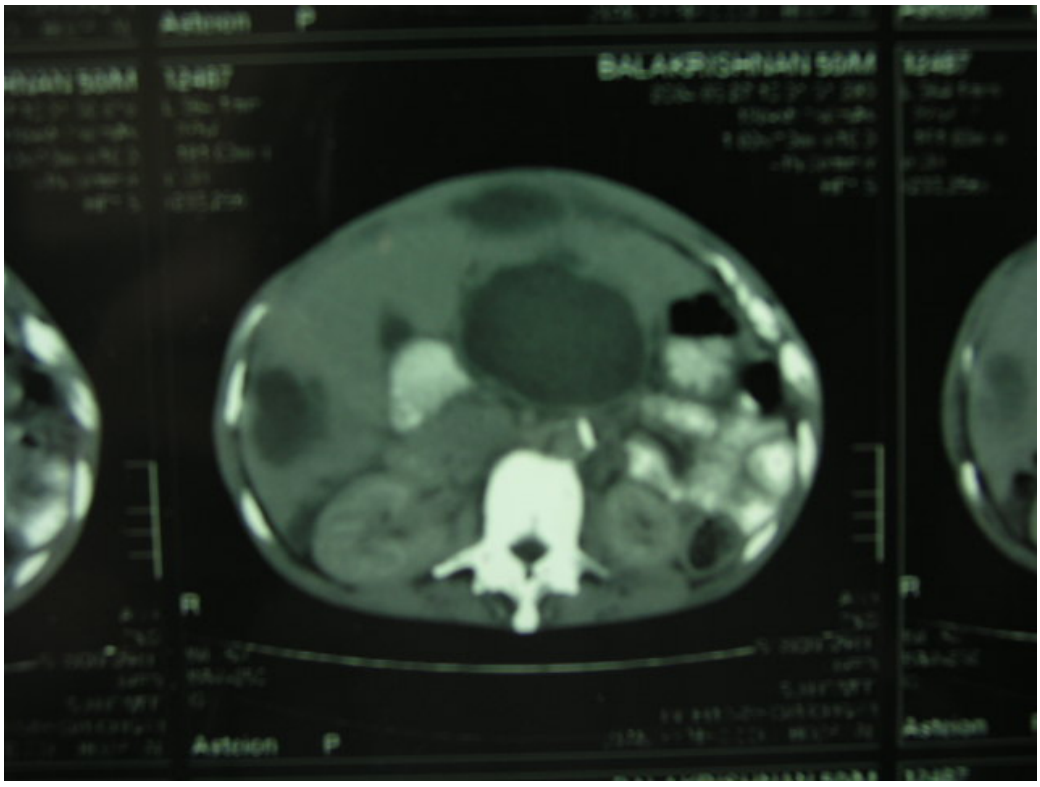
LIFE CYCLE OF ENTAMOEBA HISTOLYTICA

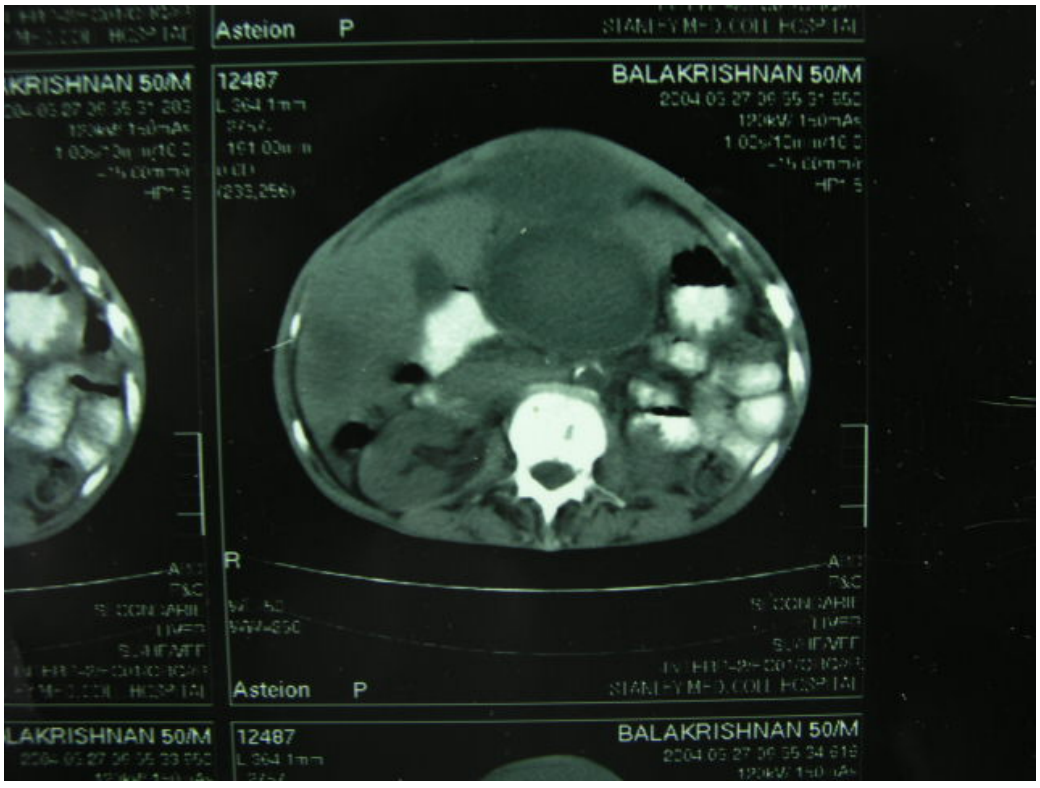


X – RAY SHOWING BASAL PNEUMONITIS WITH PLEURAL EFFUSION IN LIVER ABSCESS WITH RAISED DOME OF RIGHT DIAPHRAGM

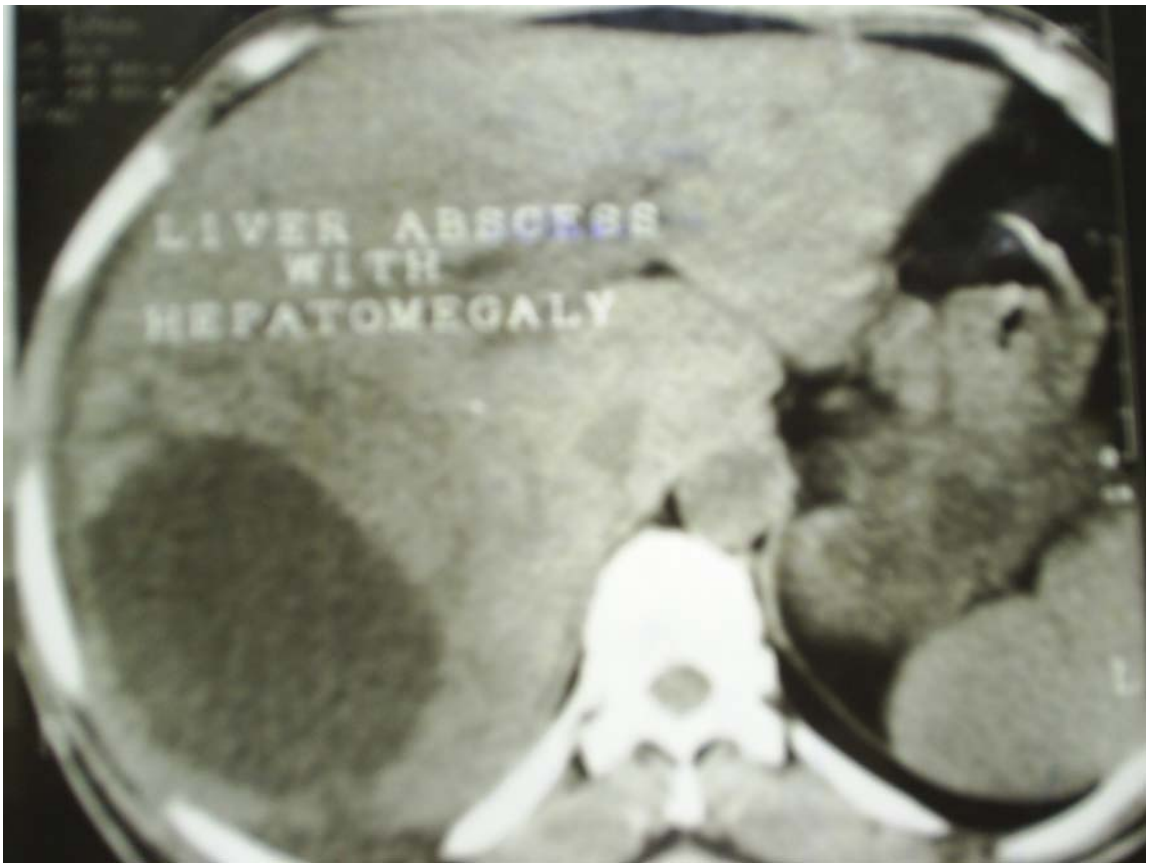


CT SCAN SHOWING MULTIPLE LIVER ABSCESS

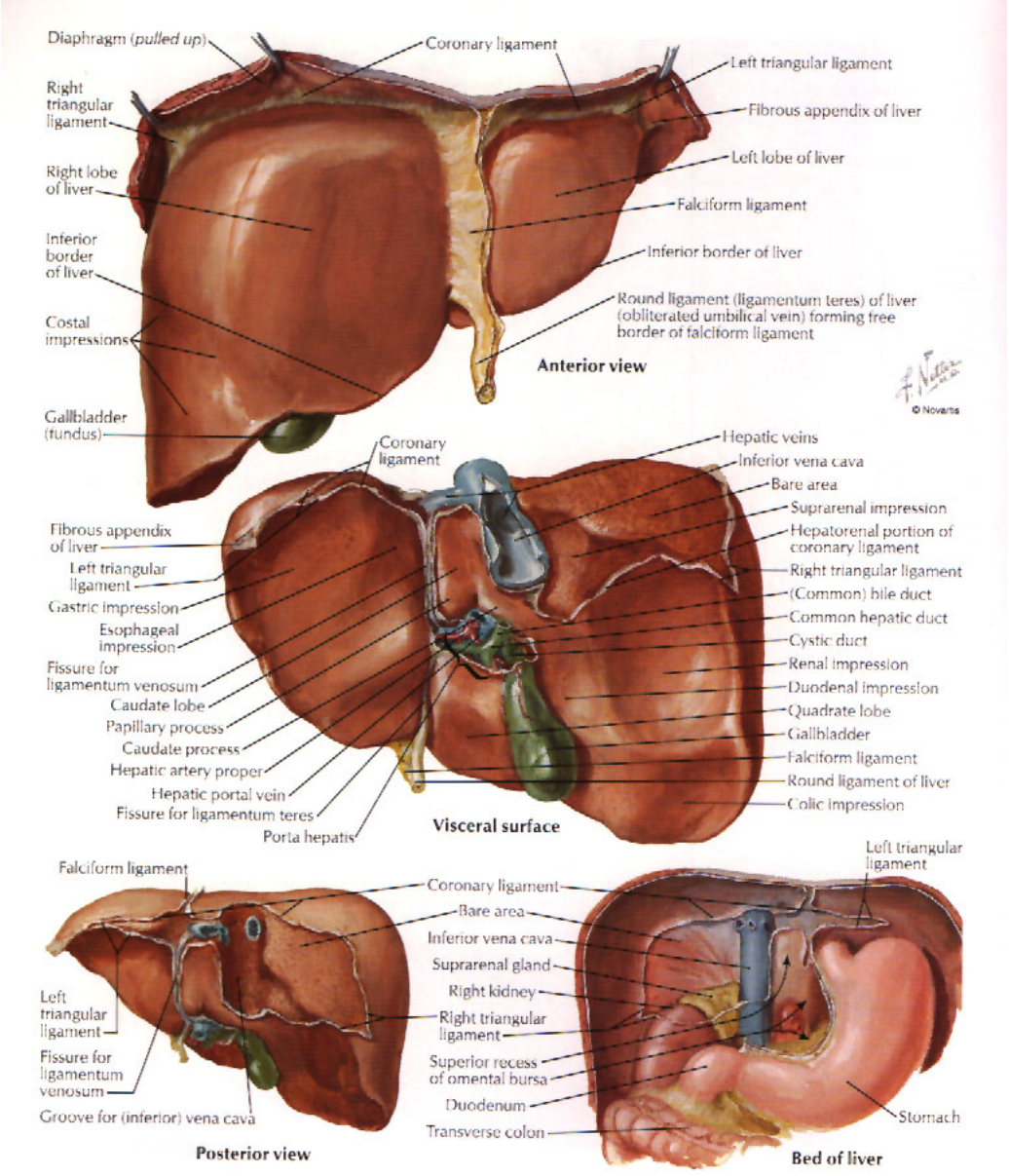




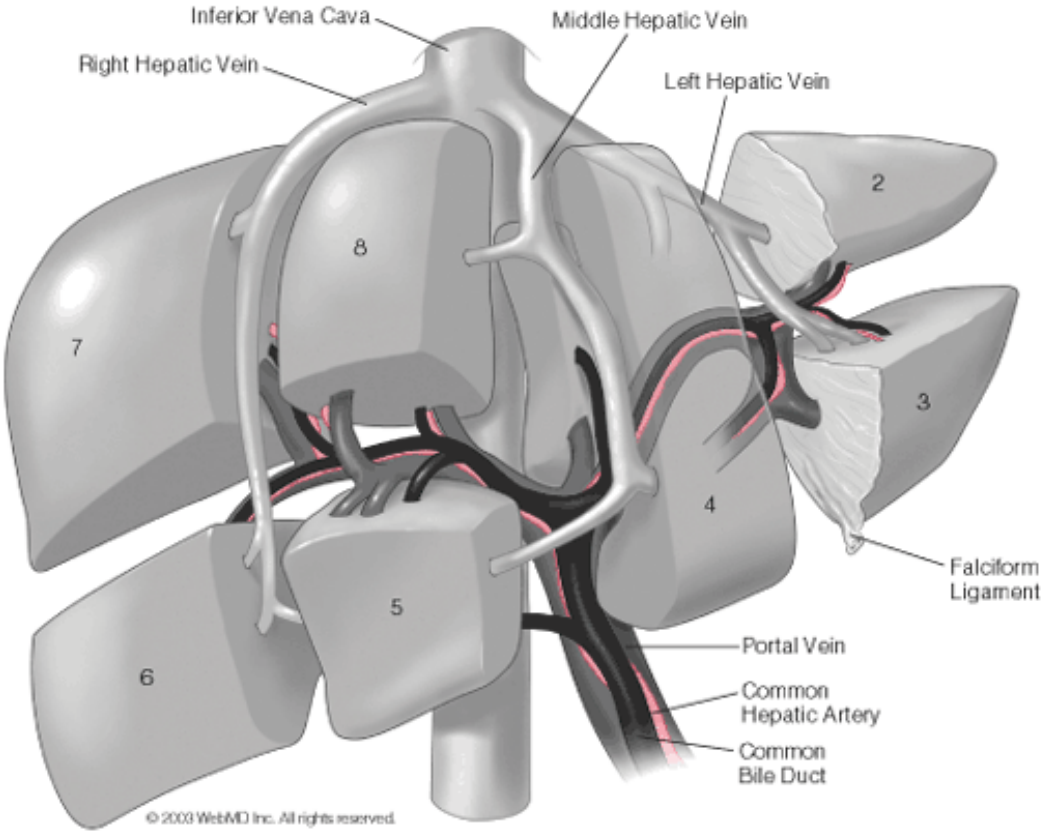
CT SCAN SHOWING RIGHT LOBE LIVER ABSCESS



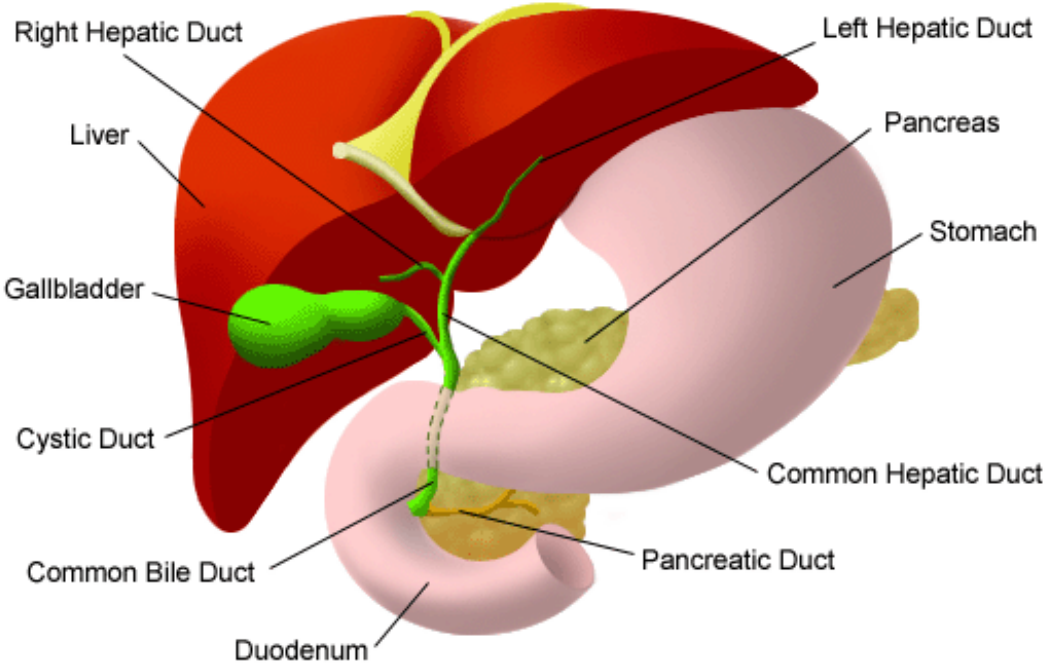
LIVER SURFACE ANATOMY



SEGMENTS OF THE LIVER – COUINAUD'S NOMENCLATURE



Biliary System

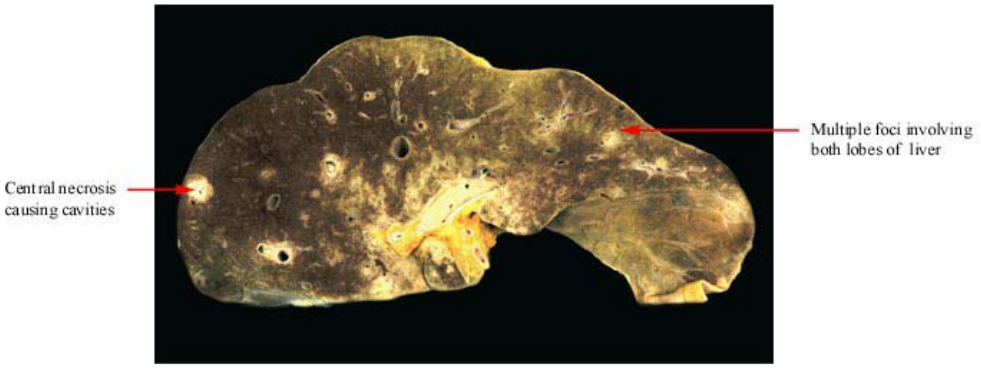


LIVER ABSCESS RUPTURING INTO PLEURAL CAVITY – INTERCOSTAL DRAINAGE

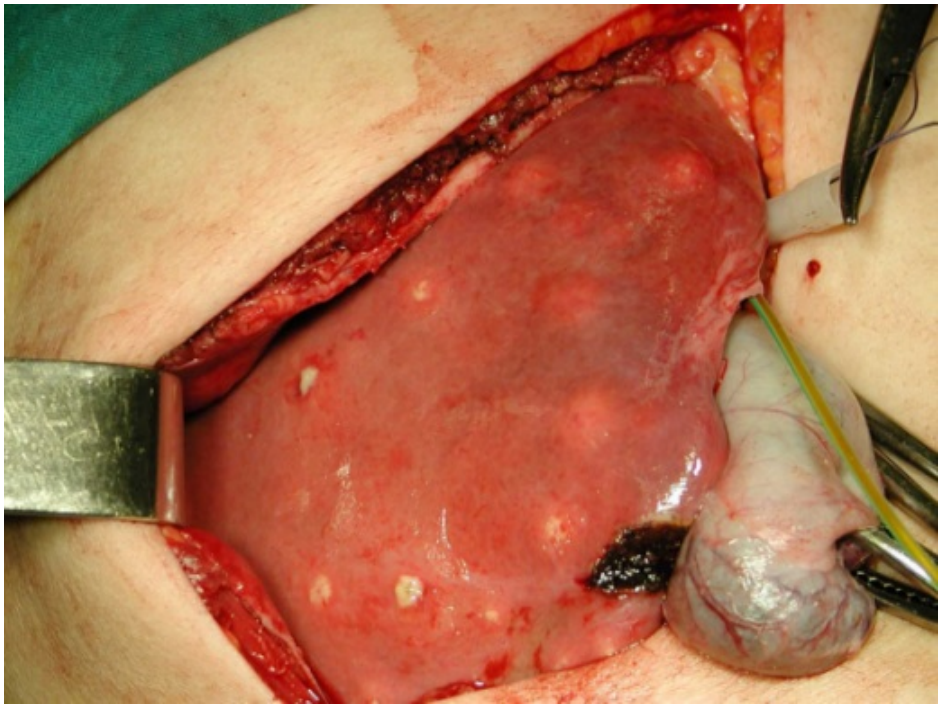
LIVER ABSCESS RUPTURING INTO PERITONEAL CAVITY – LAPAROTOMY & OPEN DRAINAGE

HUGE LIVER ABSCESS – LAPAROTOMY & OPEN DRAINAGE

SPECIMEN OF PYOGENIC LIVER ABSCESS



PYOGENIC LIVER ABSCESS

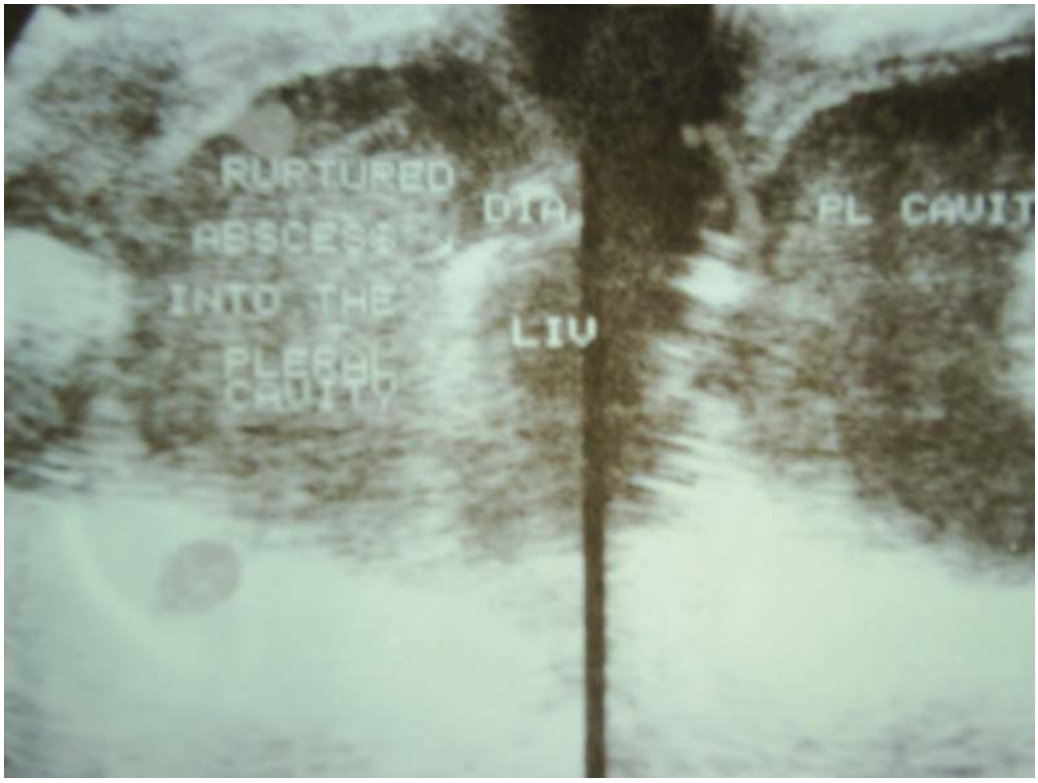


LIVER ABSCESS ASPIRATION

PUS ASPIRATED

ULTRASOUND GUIDED ASPIRATION SHOWING NEEDLE TIP

ULTRASOUND SHOWING RUPTURED LIVER ABSCESS INTO PLEURAL CAVITY



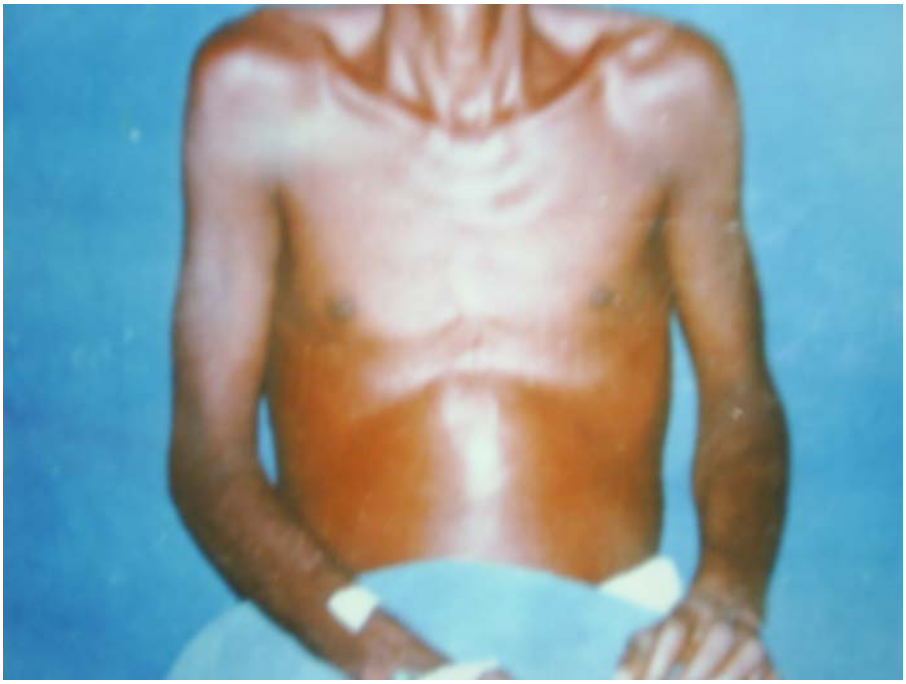
X-RAY SHOWING RUPTURED LIVER ABSCESS INTO PLEURAL CAVITY

ULTRASOUND SHOWING RIGHT LOBE LIVER ABSCESS

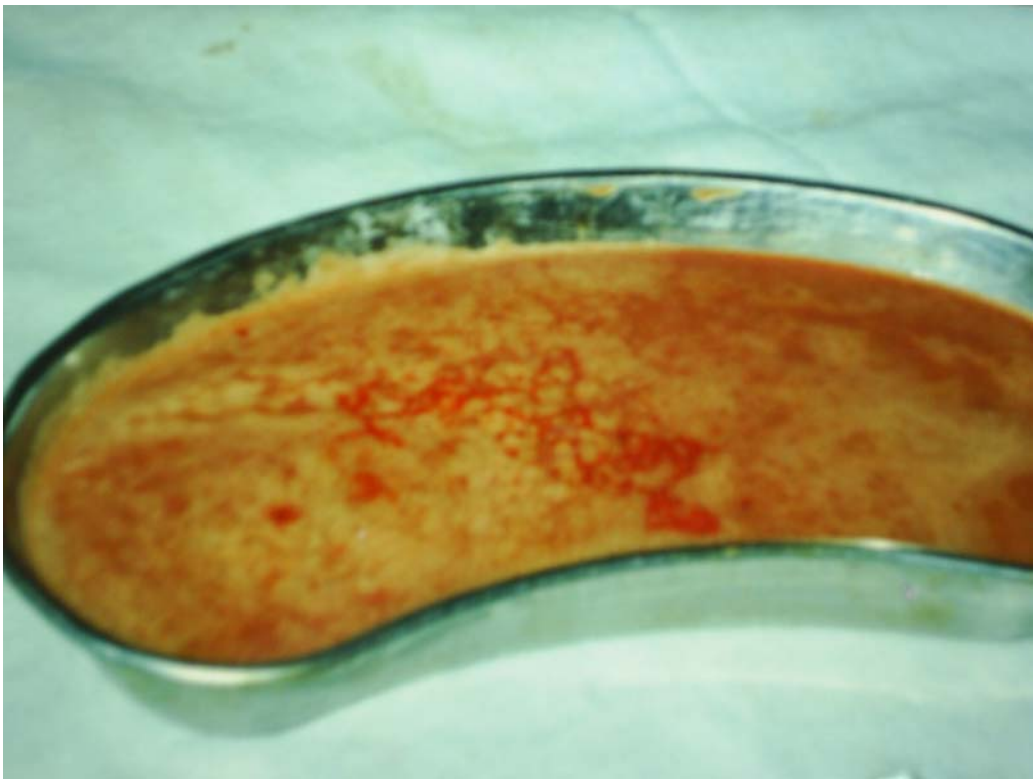
LIVER ABSCESS RUPTURING INTO PERITONEAL CAVITY

A CASE OF LEFT LOBE LIVER ABSCESS

A CASE OF LIVER ABSCESS



“ANCHOVY – SAUCE” PUS



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